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Supreme Court, U.S.
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90-No 1864

IN THE

Supreme Court of the United States

October Term, 1990

NEW YORK STATE DEPARTMENT OF HEALTH,

Petitioner,

against

JOANNA ANDRULONIS, Individually and as Conservator of the Property of JEROME ANDRULONIS; UNITED STATES OF AMERICA; GLATT AIR TECHNIQUES, INC.; GLATT GmbH; WISCONSIN ALUMNI RESEARCH FOUNDATION, INC.; WARF INSTITUTE, INC.; RALTECH SCIENTIFIC SERVICES, INC.; RALSTON PURINA COMPANY; ELI LILLY AND COMPANY; and JOHN L. THOMPSON AND SONS COMPANY,

Respondents.

ON PETITION FOR A WRIT OF CERTIORARI TO THE UNITED STATES COURT OF APPEALS FOR THE SECOND CIRCUIT

Appendix to Petition for a Writ of Certiorari

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Index to Appendix.

	Page
1. Opinion of the United States Court of Appeals, Second Circuit, Dated January 28, 1991	A1
2. Opinion of the United States District Court for the Northern District of New York, Dated December 15, 1989	A46
3. Opinion of the United States District Court for the Northern District of New York, Dated October 3, 1984	A390
4. Order of the United States Court of Appeals for the Second Circuit Denying Petition for Rehearing, Dated March 4, 1991	A399
5. Order of the United States Court of Appeals, Second Circuit, Dated January 28, 1991, Issued as Mandate March 12, 1991	A401
6. Amended Judgment of the United States District Court for the Northern District of New York, Dated December 15, 1989	A403
7. Judgment of the United States District Court for the Northern District of New York, Dated October 18, 1989	A404

Opinion of the United States Court of Appeals, Second Circuit, Dated January 28, 1991.

ANDRULONIS v. U.S.
Cite as 924 F.2d 1210 (2nd Cir. 1991)

Joanna ANDRULONIS, Individually and as Conservator of the Property of Jerome Andrulonis, Plaintiffs–Appellees/Cross–Appellants,

v.

UNITED STATES of America, Glatt Air Techniques, Inc.; Glatt GmbH; Wisconsin Alumni Research Foundation, Inc.; Warf Institute, Inc.; Raltech Scientific Services, Inc., Ralston Purina Company; Eli Lilly and Company; and John L. Thompson and Sons and Company, Defendants,

United States of America, Defendant–Appellant/Cross–Appellee.

UNITED STATES of America, Third–Party Plaintiff–Appellee,

v.

NEW YORK STATE DEPARTMENT OF HEALTH, Third–Party Defendant–Appellant.

Nos. 61, 62 and 506, Dockets 89–6274, 90–6016 and 90–6028.

United States Court of Appeals, Second Circuit.

Argued Sept. 11, 1990.

Decided Jan. 28, 1991.

Bacteriologist who contracted rabies while conducting laboratory experiment with rabies viral strain brought personal injury action along with wife against New York Department of Health, federal Government, and others. The United States District Court for the Northern District of New York, Howard G. Munson, J., 724 F.Supp. 1421, entered judgment for bacteriologist. Cross appeals were taken. The Court of Appeals, George C. Pratt, Circuit Judge, held that: (1) failure of federal government scientist to warn bacteriologist of danger of working with rabies viruses rendered Government liable; (2) remand was required for redetermination of allocation of settlement; and (3) award of $2,417,238 for bacteriologist's future custodial care was not clearly erroneous.

Reversed in part and remanded and affirmed.

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James D. Featherstonhaugh, Albany, N.Y. (Roemer & Featherstonhaugh, John R. Mineaux, of counsel), Albany, N.Y., for plaintiffs-appellees/cross-appellants.

Before PRATT, MAHONEY and WALKER, Circuit Judges.

GEORGE C. PRATT, Circuit Judge:

Jerome Andrulonis contracted the disease of rabies while conducting a laboratory experiment with a rabies viral strain. He became tragically ill and eventually suffered serious and permanent brain damage, leaving him with the mental capacity of a four-year-old. He also suffered marked personality and behavioral changes as a result of dementia secondary to rabies encephalitis. Andrulonis's mental incapacity and emotional instability are permanent. The experiment, which was conducted under the supervision of Dr. John G. Debbie, a scientist employed by the New York State Department of Health ("NYSDOH"), and observed by Dr. George M. Baer, a federal government scientist, took place in the state-operated Griffin Laboratories just outside Albany, New York.

Joanna Andrulonis, individually, and as conservator of the property of Jerome Andrulonis, her husband, brought this action against the United States of America and a number of nongovernmental defendants to recover damages for the severe injuries they have suffered because of the rabies.

The claim against the United States was, of course, brought under the Federal Tort Claims Act, 28 U.S.C. §§ 1346, 2671–2680 ("FTCA" or "the act"). The United States cross-claimed against the nongovernmental defendants and filed a third-party complaint for contribution against the State of New York. *See* N.Y. Civ.Prac.L. & R. §§ 1401–1404. Before trial, the district court dismissed Joanna Andrulonis's individual claims against the United States for failure to timely file the administrative claim required by the FTCA. *Andrulonis v. United States*, No. 79–CV–847, slip op. at 7 (N.D.N.Y. March 8, 1984); *see* 28 U.S.C. § 2675(a) and § 2401(b).

Before the end of trial, plaintiffs, through a series of agreements, had settled with all of the nongovernmental parties for a total of $1,300,000. The settling parties agreed that $225,000 would be paid in satisfaction of the primary claims of Jerome Andrulonis and that $1,075,000 would be paid in satisfaction of the derivative claims of Joanna Andrulonis.

After a nonjury trial of the remaining unresolved FTCA claims of plaintiff Jerome Andrulonis against the United States and the contribution claims of the United States against NYSDOH, Judge Munson found the government liable in negligence for Jerome Andrulonis's injuries and also found NYSDOH liable to the government on its contribution claim. In addition, con-

sidering the relative culpabilities of the settling private defendants, he found Eli Lilly and Company and John L. Thompson & Sons and Company responsible to the extent of 5 percent and exonerated the other settling defendants. The court assessed damages in the amount of $6,424,641, including $2,417,238 for the future custodial care of Jerome Andrulonis, and apportioned them 30 percent to the United States, 65 percent to NYSDOH, and 5 percent to Lilly/Thompson. *Andrulonis v. United States*, 724 F.Supp. 1421 (N.D.N.Y. 1989).

Under section 15–108 of New York's General Obligations Law, the United States was entitled, with respect to each settlement, to a setoff for the greater of "(a) the amount stipulated in the settlement agreement, (b) the consideration paid for the release, or (c) the amount of the settling defendant's equitable share of the damages." N.Y.Gen.Oblig.Law § 15–108. As noted earlier, Jerome Andrulonis had been allocated $225,000 of the proceeds of a settlement agreement with the private defendants. Of this amount, Lilly/Thompson paid $100,000 and the other settling private defendants paid $125,000. Since defendants Lilly/Thompson had settled for an amount that was less than their equitable share, the district court reduced the award by 5 percent or $321,232, representing the proportionate culpability found against Lilly/Thompson. Since the district court

found no culpable conduct attributable to the other settling defendants, it reduced Jerome Andrulonis's award by the actual dollar amount paid to him by those defendants—$125,000. This left the United States primarily responsible to Jerome Andrulonis for $5,978,409, subject to recovering from the state 68.42 percent (65/95ths) of that amount, or $4,090,427, on its third-party contribution claim. N.Y.Civ.Prac.L. & R. § 1402.

BACKGROUND

Given the complexity of this case, we must delve with considerable detail into the circumstances surrounding Andrulonis's illness and the experiment that caused it. It is unfortunate that such a long and technical narrative must follow, but a full understanding of the district court's decision cannot be reached without careful consideration of the virus used, the disease it causes, and the methods of researching rabies used at the time the tragedy occurred. The following facts include those that the district court found rabies experts reasonably should have known about the disease. Since the district court's findings are not clearly erroneous, we must accept them as true. Fed.R.Civ.P. 52(a).

Jerome Andrulonis was a 34-year-old senior bacteriologist employed to conduct rabies research in NYSDOH's Griffin Laboratory under the supervision of Dr. John G.

Debbie, a research scientist employed by NYSDOH as head of the rabies labs at Griffin. Dr. Debbie was persuaded to conduct rabies research by Dr. George M. Baer, Chief of the Viral Zoonosis Branch and Rabies Laboratory at the Center for Disease Control ("CDC"), a division of the United States Department of Health and Human Services. The research being done by Dr. Debbie and Andrulonis was part of a joint effort by the NYSDOH and the CDC to develop a technique for mass immunization of wildlife in the hope of eradicating the incidence of rabies transmitted to man.

To realize this goal, Dr. Debbie was working on an oral vaccine that could be distributed to wildlife. Because the initial virus strains tested by Dr. Debbie could not withstand the destructive stomach acids of some species, he decided, upon the advice of Dr. Baer, to try a method called "enteric coating." Under this method, a pill, or nonpareil, would be coated first with the rabies virus, then a protective coating, and finally, a shell that could withstand stomach acids but would disintegrate in the animal's intestines, allowing the virus to be absorbed there. The virus would not cause development of the disease, but would cause the body to produce antibodies that would protect the animal against subsequent exposures to the rabies virus.

Dr. Debbie contacted the coating department at defendant WARF Institute, Inc. ("WARF"), which on March 30, 1976, sent a

representative to demonstrate a coating technique using a Uni–Glatt machine, manufactured by defendant Glatt GmbH and distributed by defendant Glatt Air Techniques, Inc. ("the Glatt defendants"). The Uni–Glatt machine operated by suspending nonpareils in a column of upflowing air confined in a tube. The suspended nonpareils would then be coated with aerosolized virus.

When the machine operated, the aerosol itself could be seen inside the tube, although any leakage into the atmosphere was not visible to the unaided eye and left no trace outside the machine. However, nonpareils would occasionally leak out of the machine into the lab environment, and the final coating process created a visible reddish dust that escaped from the machine leaving the entire laboratory coated with the reddish dust after each run. During the initial demonstration run, it became apparent to everyone in the laboratory that the machine was not airtight. Despite this, the only precautions required of the lab workers were the wearing of a mask and gloves, and the maintenance of a high level of rabies antibodies in their blood systems through periodic inoculations with a rabies vaccine manufactured by defendant Eli Lilly and Company and distributed by defendant John L. Thompson & Sons and Company ("Lilly/Thompson").

Funding for the Uni–Glatt rabies experiments was provided by the federal CDC. In exchange, Dr. Debbie was to provide the

CDC with some of the coated nonpareils prepared in the course of the Uni-Glatt experiments. Beyond providing the funding, CDC officials made no attempt to direct the day-to-day operations of the project, to devise any protocol, or otherwise to control the research efforts at Griffin.

Dr. Debbie used the Uni-Glatt in two more experiments in July and September of 1976, both times using a rabies viral strain prepared by Andrulonis with a titer of 6. "Titer" is the term used to indicate a standardized measure of a substance's ability to produce a defined result. With respect to the rabies virus, the titer measures the ability to kill laboratory mice after intracerebral injection of a solution containing that virus. The titer is expressed exponentially.

During the July and September runs, heat generated by the machine killed most of the viral particles and reduced the titer of the vaccine to 1, a level that produced irregular serum antibody levels. To overcome this problem, Dr. Debbie asked Dr. Baer to prepare a viral strain with a higher titer.

Dr. Baer, in keeping with CDC's general practice of preparing and supplying viral strains to many private and state-operated labs, prepared a relatively unknown viral strain, ERA-BHK/21, with a titer of 8.1. This was derived from a commercially available ERA vaccine, but it had a much higher concentration of viral particles and was less susceptible to heat. While the basic ERA vaccine had several known fa-

vorable characteristics, the particular characteristics of ERA-BHK/21 were relatively unknown to most scientists in March of 1977, since it was several times removed from the commercial ERA vaccine. ERA-BHK/21 was created through a process called "passaging" that had the potential for producing unpredictable changes in characteristics. The handful of studies on ERA-BHK/21 had reported mixed results about pathogenicity, and no one knew with certainty whether man was susceptible to developing the disease of rabies as a result of infection from this ERA-derived virus strain. The CDC generally recommended, as a precaution, that the virus be treated as pathogenic to man.

Several other factors, in addition to uncertainty about the characteristics of ERA-BHK/21, complicated the situation. People usually contract rabies through the saliva of an infected animal during a bite. Infection through aerosol exposure, the route of infection in Andrulonis's case, is much more rare and was not even recognized until the late 1950s or early 1960s. Not all exposures lead to infection; not all infections lead to the disease. Regardless of the route of infection, the chances of getting the disease after exposure are closely related to the quantity of viral particles, or the dose, to which a person is exposed. When the exposure is aerosol in nature, the dose is probably the single most important factor in determining whether the virus will cause the disease.

In addition, the pathogenesis of the disease is very different between the airborne and the bite routes. With a bite, the area is bathed with blood, so if the bitten person has been immunized, the antibodies in the blood have an opportunity to neutralize the virus before it can invade the central nervous system. In contrast, when airborne, the virus hits olfactory nerves that are exposed to the environment and that have as their only protection the nasal mucosa. Ordinarily, olfactory nerves do not have significant contact with antibodies in the blood stream. Thus, even if a person has been immunized, the antibodies do not have a chance to come into contact with the infected nerves, unless the vaccine is able to produce antibodies in the mucosa.

In 1976–1977, when Andrulonis was exposed to the virus, it was not known whether the Lilly vaccine he and the other lab technicians were using could protect against aerosol exposure. Lilly did not conduct studies even after becoming aware of the possibility of aerosol exposure, nor did it warn users of the potential that its vaccine might not protect against aerosol exposures. In light of the meager then-existing knowledge about aerosol exposures and vaccines, the district court found that safe lab practices mandated avoidance of all aerosol exposures and that virus-laden aerosols should not even be created unless absolutely necessary for the research goal, and in those cases, the aerosols should be physically contained using one of the readily-available containment systems.

On the evening of March 28, 1977, Dr. Baer personally delivered the specially prepared ERA-BHK/21 virus strain with a titer of 8.1 to Dr. Debbie at Griffin. He informed Dr. Debbie of the titer and Dr. Debbie relayed this information to the other lab workers the next morning. No disclosure was made about how the strain was prepared or how many times it was passaged. No safety inspection was made of the lab premises, but this was consistent with ordinary CDC practice in similar circumstances.

The tragic experiment took place the next day, March 29th. With Dr. Baer observing, Dr. Debbie started the machine and began circulating the nonpareils in the glass tube. The aerosolization process using the ERA-BHK/21 virus lasted over an hour, during which time both Dr. Debbie and Andrulonis worked in close proximity to the machine. The process required close attention, and usually Andrulonis or Dr. Debbie sat near the machine and tapped the glass tube with the handle of a screwdriver to prevent nonpareils from sticking together. Commonly, Andrulonis would place his face within six to twelve inches of the tube while tapping it, and would remain that close to the machine for minutes at a time throughout the run. Dr. Baer also observed the process closely and, at times, was within a foot of the machine.

Dr. Baer left on the evening of March 29th. Another coating run was done the next day, but this time using a viral strain prepared by Andrulonis that had a titer of 5.5.

The district court found on adequate, albeit disputed, evidence that Andrulonis had contracted rabies during the March 29, 1977, experiment that Dr. Baer observed. To begin with, Andrulonis's only contact with the virus was at work. On the job, it was unlikely that he got rabies from routine procedures; more likely, he contracted the disease from an "unusual" exposure. Of his four possible exposures between March of 1976 and March of 1977, compelling evidence points to the March 29, 1977, experiment as the cause of his illness, which first appeared in April of 1977. As to the other exposures, in March, July and September of 1976, a six-month incubation period is within the realm of possibility, but the average is 20–60 days. Only 14 percent of the incidents of rabies have an incubation period greater than 90 days regardless of the method of exposure. Since this was an aerosol exposure, the incubation period was more likely to be shorter than longer.

Another unusual aspect of the March 29th experiment was its use of the most highly concentrated virus with a titer of 8.1. This means there were 100 times more viral particles than a 6.0 titer and 400 times more viral particles than a 5.5 titer.

Further, the viral strain used in the March 29th experiment was more resistant to the heat generated by the machine. Finally, the district court accepted the opinion testimony of various experts who stated with a reasonable degree of medical and scientific certainty that Andrulonis contracted the disease of rabies as a result of exposure in the March 29th experiment.

The district court found the government liable under New York law on three independent theories of negligence: supplier's duty to warn, non-negligent creation of a hazard, and negligent entrustment. The government challenges each of these holdings. The district court also held that the discretionary function exception of the Federal Tort Claims Act was inapplicable to this case, and both the government and NYSDOH appeal from this holding.

In addition, the government and NYSDOH appeal from the district court's approval of the allocation of settlement proceeds; NYSDOH appeals the district court's apportionment of damages; and finally, Jerome Andrulonis cross-appeals from the court's determination of the cost of future custodial care.

DISCUSSION

We first turn to the question of whether the discretionary function exception protects the government from suit in these circumstances. If the exception were to apply in this case, it would bar this action against the government, and we could dismiss the complaint on that ground alone. However, since we determine that the exception is inapplicable here, we must then discuss the question of whether New York law imposes liability on the government under the circumstances of this case. Of the three theories relied on by the district court, we determine that the failure to furnish an adequate warning is sufficient to hold the government liable, so we need not reach the merits of the other two theories.

We next will address the questions of whether the settlement proceeds were properly allocated between Joanna and Jerome Andrulonis, and whether the district court properly apportioned the percentages of responsibility between the federal government and the state. Finally, we will discuss the plaintiff's cross-appeal on whether the district court correctly calculated his damages for future custodial care.

A. *Discretionary Function Exception*

[1] Initially, we take up whether this action was barred by the discretionary function exception to the FTCA. The tort

claims act generally authorizes suits against the United States for damages

> for injury or loss of property, or personal injury or death caused by the negligent or wrongful act or omission of any employee of the Government while acting within the scope of his office or employment, under circumstances where the United States, if a private person, would be liable to the claimant in accordance with the law of the place where the act or omission occurred.

28 U.S.C. § 1346(b). The act further provides that the United States shall be liable with respect to tort claims "in the same manner and to the same extent as a private individual under like circumstances." 28 U.S.C. § 2674.

Although the act waives the sovereign immunity of the United States, it does not do so in all respects. Congress carefully excepted several important classes of tort claims from the act's broad waiver of immunity. Of particular relevance here, section 2680(a) provides that the act shall not apply to

> [a]ny claim based upon • • • the exercise or performance or the failure to exercise or perform a discretionary function or duty on the part of a federal agency or an employee of the Government, whether or not the discretion involved be abused.

28 U.S.C. § 2680(a). "The discretionary function exception • • • marks the bound-

ary between Congress' willingness to impose tort liability upon the United States and its desire to protect certain governmental activities from exposure to suit by private individuals." *United States v. S.A. Empresa De Viacao Aerea Rio Grandense (Varig Airlines)*, 467 U.S. 797, 808, 104 S.Ct. 2755, 2761–2762, 81 L.Ed.2d 660 *rehearing denied*, 468 U.S. 1226, 105 S.Ct. 26, 82 L.Ed.2d 919 (1984).

The Supreme Court in *Dalehite v. United States*, 346 U.S. 15, 27–28, 73 S.Ct. 956, 963–964, 97 L.Ed. 1427 (1953), analyzed the legislative history of the act and concluded that its waiver of sovereign immunity was inapplicable to "acts of a governmental nature or function." The basis for the exception was congress's wish "to prevent judicial 'second-guessing' of legislative and administrative decisions grounded in social, economic, and political policy through the medium of an action in tort." *Varig Airlines*, 467 U.S. at 814, 104 S.Ct. at 2764–2765. The foundation for this exception is protection of "the principles embodied in the separation of powers doctrine by keeping the judiciary from deciding questions consigned to the executive and legislative branches of the government." *Caban v. United States*, 671 F.2d 1230, 1233 (2d Cir.1982).

> By barring tort liability for activities that require the alleged tortfeasor to consider and weigh competing policies in arriving at his decision, this test protects courts from "involve[ment] in making ... decision[s] entrusted to other branches of the government," and especially from questions involving "not negligence but social wisdom, not due care but political practi-

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> cability, not reasonableness but economic expediency".

Id. (citations omitted). Congress thus sought to assure that tort liability would not "seriously handicap efficient government operations." *United States v. Muniz*, 374 U.S. 150, 163, 83 S.Ct. 1850, 1858, 10 L.Ed.2d 805 (1963).

Determining whether specific conduct is protected by the discretionary function exception to the FTCA is sometimes difficult. The act itself contains no definition of the types of discretionary functions within the exception. "The principal difficulty is simply that all federal employees exercise a certain amount of discretion in the discharge of their responsibilities." *Caban*, 671 F.2d at 1232.

[2] To determine whether conduct falls within the exception, we must focus on "the nature of the conduct, rather than the status of the actor," *Varig Airlines*, 467 U.S. at 813, 104 S.Ct. at 2764, and use a two-step process to examine the challenged conduct. *See Berkovitz v. United States*, 486 U.S. 531, 536, 108 S.Ct. 1954, 1958–1959, 100 L.Ed.2d 531 (1988); *In re Joint E. & So. District Asbestos Litigation*, 891 F.2d 31, 36 (2d Cir.1989). First, we must consider whether the conduct was a matter of choice for the acting employee, and if so, we must then determine whether the employee's actions involved public policy considerations. *Asbestos Litigation*, 891 F.2d at 36.

[3] As to the first step, the Supreme Court has determined that "conduct cannot

be discretionary unless it involves an element of judgment or choice." *Berkovitz*, 486 U.S. at 536, 108 S.Ct. at 1958–1959. Thus, when a federal statute, regulation, or policy specifically dictates a course of action for an employee to follow, the employee has no option but to adhere to the directive. *Id.* If the employee can make no choice, then there can be no discretion for the exception to protect. *Id.* No such directive was applicable to Dr. Baer in this case; at the critical points in these events he had the power to choose his actions.

But this does not end the inquiry. As we have noted, to qualify for the exception, Dr. Baer's critical choices had to involve policy considerations, and here they did not. The *Berkovitz* Court stated that "[t]he exception, properly construed, * * * protects only governmental actions and decisions based on considerations of public policy." *Id.* at 537, 108 S.Ct. at 1959. If an action leaves "room for policy judgment and decision there is discretion." *Dalehite*, 346 U.S. at 36, 73 S.Ct. at 968.

In *Indian Towing Co. v. United States*, 350 U.S. 61, 76 S.Ct. 122, 100 L.Ed. 48 (1955), the Court distinguished between an initial government decision to build a lighthouse and subsequent actions to maintain the lighthouse. The initial decision to undertake and maintain a lighthouse service was a discretionary judgment; however, the failure to adequately maintain the lighthouse in good condition, since it did not involve any permissible exercise of policy judgment, subjected the government to suit under the FTCA. *Id.* at 69, 76 S.Ct. at 126–127; *see also Berkovitz*, 486 U.S. at 538 n. 3, 108 S.Ct. at 1959 n. 3.

Similarly, in *Caban*, we determined that the adoption of a regulation by the INS was discretionary but a wrongful detention by an INS employee was not discretionary. *Caban*, 671 F.2d at 1233. *See also Caraballo v. United States*, 830 F.2d 19, 21–22 (2d Cir.1987) (initial decision to patrol a national park was discretionary, but the manner in which decision was executed was not discretionary); *Eklof Marine Corp. v. United States*, 762 F.2d 200, 204–05 (2d Cir.1985) (initial decision to provide navigational aid by marking obstructions was discretionary, but the precise manner of marking an obstruction did not implicate policy and was not a discretionary act).

The government tries to distinguish this line of cases in two ways. First, it contends that the *Indian Towing* approach is inapplicable because the government here did not decide upon a particular safety measure and then negligently implement its decision. The government would thus restrict *Indian Towing* to cases involving a decision to provide safety measures. We decline to limit *Indian Towing* so narrowly. We think that case rested not so much on the government's decision to install a safety device, a lighthouse, but on its negligent maintenance of the lighthouse and the fact that decisions to maintain the lighthouse did not implicate policy considerations. In *Indian Towing*, only the initial decision to install the lighthouse was discretionary.

[4] Sometimes, even the execution of a policy decision is subject to protection by the discretionary function exception; this occurs when the steps in the execution process also require policy judgments. For example, in *Varig Airlines*, the Court not only found that the Federal Aviation Administration's implementation of a program for compliance review was discretionary, but it also found that the acts of the FAA employees who executed the program were protected, because those employees "were specifically empowered to make policy judgments regarding the degree of confidence that might reasonably be placed in a given manufacturer, the need to maximize compliance with FAA regulations, and the efficient allocation of agency resources." *Varig Airlines*, 467 U.S. at 820, 104 S.Ct. at 2767–68. However, in *Caban*, we determined that even if regulations and statutes appear to vest immigration officials with broad discretion, where the language goes only to the standard of care by which governmental employees' behavior is to be judged, the language does not convert the discharge of prescribed responsibilities into decisions which involve a choice between competing policy considerations. *Caban*, 671 F.2d at 1233. In short, the key inquiry is whether the decisions, be they initial decisions or implementation decisions, were necessarily susceptible to policy analysis. *Asbestos Litigation*, 891 F.2d at 37; *United States Fidelity & Guar. Co. v. United States*, 837 F.2d 116, 121 (3d Cir.), *cert. denied*, 487 U.S. 1235, 108 S.Ct. 2902, 101 L.Ed.2d 935 (1988).

In this case, the government's decisions to encourage rabies research, provide funding for the research, and provide NYSDOH with the virus were all decisions that implicated policy considerations and therefore were protected by the discretionary function exception. The same is not true, however, with Dr. Baer's alleged failure to warn of the extreme dangers presented by the particular circumstances of the March 29th experiment which Dr. Baer failed to interrupt. The situation simply did not lend itself to policy balancing, not is there any indication that Dr. Baer considered the policy implications or the pros and cons of allowing the experiment to proceed. Once he became aware of the risks, Dr. Baer was obligated to give warning. His is precisely the type of negligent omission for which congress waived the government's sovereign immunity in order to provide redress to citizens for the torts of government actors.

The government relies on *Dalehite* to support its contention that even mundane decisions necessary to execute a policy decision are entitled to protection under the discretionary function exception. *Dalehite* involved a tragic explosion of fertilizer that had been manufactured and shipped at the instigation and under the supervision of the government. The Supreme Court held that even those actions that were necessary in the execution of a policy decision were entitled to protection under the discretionary function exception. *Dalehite*, 346 U.S. at

36, 73 S.Ct. at 968. *Dalehite* is distinguishable, however, because the Court could not find there any specific instance of negligence by an employee. The accident was "a complex result of the interacting factors of mass, heat, pressure and composition," *id.* at 42, 73 S.Ct. at 971, and no single action was identified as the cause of the accident. Liability of the government thus could only have been based on the larger policy decision to manufacture and store the fertilizer.

The government suggests that a similar inability to pinpoint the precise source should prevent liability in this case, because this accident, too, was the result of complex processes. The district court, however, was able to penetrate the complex facts of this case and determine that Jerome Andrulonis's illness resulted, at least in part, from Dr. Baer's failure to warn of the potentially disastrous consequences of the March 29th experiment he was observing. This was a nondiscretionary negligent omission that did not implicate any balancing of policy factors. The district court's findings on this point are not clearly erroneous, and the government is therefore not entitled to protection from suit under the discretionary function exception.

B. *Supplier's Duty to Warn*

The district court found the government liable on three independent theories of tort liability. We need discuss only the supplier's duty to warn, because it is sufficient to affirm on the liability issue.

Under the FTCA, the law of the state where the tort occurred applies. 28 U.S.C. § 1346(b); *Hatahley v. United States*, 351 U.S. 173, 182, 76 S.Ct. 745, 752, 100 L.Ed. 1065 (1956); *Montellier v. United States*, 315 F.2d 180, 185 (2d Cir.1963). Because most of the acts or omissions of Dr. Baer occurred in New York, the substantive law of New York governs this case.

[5, 6] Under established New York law, a supplier of a product which the supplier knows or should know is dangerous if used in the usual and expected manner has a duty to adequately warn users of the product of the danger unless the danger is obvious or well-known. *Young v. Elmira Transit Mix, Inc.*, 52 A.D.2d 202, 204–05, 383 N.Y.S.2d 729, 731 (4th Dept.1976) (Cardamone, J.) (citing Restatement (Second) of Torts § 388); *Billiar v. Minnesota Mining and Mfg. Co.*, 623 F.2d 240, 243 (2d Cir. 1980). *See also Schumacher v. Richards Shear Co., Inc.*, 59 N.Y.2d 239, 246–47, 464 N.Y.S.2d 437, 441, 451 N.E.2d 195 (1983). Breach of this duty can lead to liability in negligence.

The government initially argues that a supplier's duty to warn is inapplicable to this case because the doctrine should be limited to a commercial context; that purely scientific activities should be exempt from the rigors of traditional tort law; that the risk-spreading rationale underlying imposition of this duty should not be applicable because the government is not an income-producing entity; and that imposing liability in this situation would tend to curtail scientific research, and restrict the flow of information and exchange of knowledge which are vital for producing health-related benefits.

Although we are sympathetic to the government's policy arguments, they simply are not supported by the applicable law. In the first place, the FTCA specifically provides for the imposition of liability on the federal government as if it were a private person. The fact that the government does not generate income and is not considered a commercial entity is irrelevant in light of congress's decision that governmental immunity is waived in certain circumstances if government employees commit torts.

[7] Furthermore, we agree with the district court that New York does not require a commercial transaction as a prerequisite to liability for breach of a duty to warn. Restatement (Second) of Torts § 405; *Pease v. Sinclair Refining Co.*, 104 F.2d 183, 186 (2d Cir.1939) (applying New York

law). In addition, and contrary to the government's contention, there is probably no entity better able to spread the risk of scientific research than the government, which has the biggest "customer" base of all—taxpayers. Finally, in a situation where our entire population may benefit from eradication of rabies, it is not unreasonable to have the potential beneficiaries share the cost of an unfortunate incident occurring in the search for an effective method for vaccination against this lethal disease.

Although the imposition of a duty to warn of potential hazardous consequences may, as the government argues, tend to chill the collegial atmosphere of the scientific community, we think that the benefits of imposing such a duty, at least in the highly unusual circumstances of this case, far outweigh any potentially chilling effects. All scientists should assure that reasonable and appropriate safety precautions are taken in the lab. Laboratory staffs should not have to bear the financial burdens in addition to the personal risks that accompany the quest for scientific advancement. If identifiable negligence does occur, its economic consequences need not be excused as necessary for the benefit of science, no matter how noble the goals of the supervising scientists.

[8] The government also contends that even if the imposition of a supplier's duty to warn is the appropriate legal standard,

Dr. Baer satisfied this duty by informing Dr. Debbie of the extremely high concentration of the virus. The district court found that at the time Dr. Baer gave the warning, it was sufficient merely to advise Dr. Debbie that the viral strain was highly concentrated; given what Dr. Baer knew at that time, the warning was proportionate to the risk. However, the district court also found that when Dr. Baer actually observed the experiment, he should have realized the extreme danger created by using his ERA-BHK/21 virus in a leaky aerosolizing machine and should have immediately supplemented his warning. The government argues that requiring Dr. Baer to supplement the warning just because he was a rabies expert and he decided to stay at Griffin to observe the experiment is incorrect.

[9, 10] Under the applicable New York law, we disagree. In *Cover v. Cohen*, 61 N.Y.2d 261, 274-75, 473 N.Y.S.2d 378, 385, 461 N.E.2d 864 (1984), the New York Court of Appeals determined that a manufacturer or retailer may incur liability for failing to warn of newly discovered dangers in the use of a product that come to his attention after manufacture or sale. These dangers may be discovered through advancements in the state of the art, with which the manufacturer is expected to stay abreast, or through being made aware of later accidents involving dangers in the product, of which warning should be given to users.

Although a product may be reasonably safe when manufactured and sold and involve no then-known risks of which warning need be given, risks thereafter revealed by user operation and brought to the attention of the manufacturer or vendor may impose upon either or both an additional duty to warn. *Id.* at 275, 473 N.Y.S.2d at 385, 461 N.E.2d 864. The type of notice that will trigger a manufacturer's post-delivery duty to warn is a function of the degree of danger involved and the number of instances reported, *id.*, and these are questions of fact. *Id.* at 276, 473 N.Y.S.2d at 385, 461 N.E.2d 864.

In this case, Judge Munson determined that Dr. Baer, while observing the experiment in the lab, should have known of the dangers involved and that he had sufficient notice to trigger a duty to provide additional warning. Dr. Baer was observing an experiment with the extremely potent rabies virus he had supplied being used in a leaky machine in a way that could potentially cause great harm to those present in the lab. In these circumstances, he should have realized the risks and warned against continuing the experiment without additional precautions.

Judge Munson based his conclusion on several subordinate findings: (1) whenever aerosols were used at the CDC labs in Georgia, a laminar flow containment system was also used; (2) Dr. Baer knew that safe lab practice required containment; (3) Dr. Debbie told Dr. Baer about the red

dust that had escaped from the Uni–Glatt machine during the third run of the experiments; (4) Dr. Baer saw nonpareils escape from the machine; (5) Dr. Baer saw Dr. Debbie patch the machine with electrical tape; (6) photographs of the machine showed that it did not appear to be airtight; and (7) Dr. Baer saw that the machine had to be assembled by hand in the laboratory for each experiment. Judge Munson also found that Dr. Baer's testimony, denying his knowledge of the Uni–Glatt's leak, lacked credibility.

When Dr. Baer saw that the Uni–Glatt was not airtight, he should have immediately alerted Dr. Debbie and the lab staff, including Andrulonis, of the hazards posed by the way the ERA–BHK/21 virus was to be used; his failure to do so was, under New York law, a breach of duty to those in the lab, including Andrulonis.

Finally, the government argues that even if the case is properly a duty-to-warn action, and even if Dr. Baer's single preliminary warning was inadequate, the government still cannot be liable, because the case falls within the "knowledgeable user" exception. This argument focuses on proximate cause, which is a necessary predicate to finding liability. *Sheehan v. City of New York*, 40 N.Y.2d 496, 501, 387 N.Y.S.2d 92, 95, 354 N.E.2d 832 (1976).

[11] In duty to warn cases, New York recognizes two circumstances that would preclude a finding of proximate cause: obviousness and the knowledgeable user.

Proximate cause cannot be found when the dangers are obvious or well-known. *Lancaster Silo & Block v. Northern Propane Gas*, 75 A.D.2d 55, 427 N.Y.S.2d 1009, 1015 (4th Dept.1980). Nor can it be found with a knowledgeable user, one who is actually aware of the dangerous nature of the product supplied. *Belling v. Haugh's Pools Ltd.*, 126 A.D.2d 958, 959, 511 N.Y.S.2d 732, 733 (4th Dept.), *appeal denied*, 70 N.Y.2d 602, 518 N.Y.S.2d 1024, 512 N.E.2d 550 (1987). Although the government relies here solely on the knowledgeable user doctrine, loosely referred to as the "knowledgeable user exception", a brief look at "obviousness" is necessary to understand why the government's argument fails.

[12, 13] If a danger is obvious, there is no duty to warn against it. *Kerr v. Koemm*, 557 F.Supp. 283, 287 (S.D.N.Y. 1983). The Restatement (Second) of Torts § 388 and comment (k) state that a duty to inform users of a danger exists if the supplier of a product has no reason to expect that the user will discover its condition and realize the danger involved. *See also Lancaster Silo*, 427 N.Y.S.2d at 1015; *Young*, 383 N.Y.S.2d at 731; *Kerr*, 557 F.Supp. at 287. This inquiry into the obviousness of the danger depends not upon actual knowledge of the user, but upon whether the danger was sufficiently obvious that it would be unreasonable to impose a duty to warn on the manufacturer. *Kerr*, 557 F.Supp. at 287. Thus, the focus of the

"obviousness" inquiry is upon the objective reasonableness of the supplier's judgment about whether users will perceive the danger. *Id.* The danger must be so apparent or so clearly within common knowledge that a user would appreciate the danger to the same extent that a warning would provide. *Belling*, 511 N.Y.S.2d at 733. The district court held that the danger here was not obvious because Dr. Baer actually witnessed the experiment and should have immediately recognized that neither Dr. Debbie nor Andrulonis realized the hazards they were facing. On appeal the government does not challenge that conclusion.

[14] In contrast, the "knowledgeable user exception", on which the government does rely, involves a subjective test: whether the particular user was aware of the danger. *McDaniel v. Williams*, 23 A.D.2d 729, 257 N.Y.S.2d 702 (1st Dept. 1965); *Rosebrock v. General Electric Co.*, 236 N.Y. 227, 140 N.E. 571 (1923); *Kerr*, 557 F.Supp. at 287. Its rationale is that knowledge is equivalent to prior notice. *Billiar*, 623 F.2d at 243. Where the plaintiff knows of a danger, a warning cannot increase his awareness of its presence, *Kerr*, 557 F.Supp. at 286, and where a warning would not have prevented the harm, a failure to warn cannot be the proximate cause of the injury. *Torrogrossa v. Towmotor Co.*, 44 N.Y.2d 709, 711, 405 N.Y.S.2d 448, 449, 376 N.E.2d 920 (1978); *Billiar*, 623 F.2d at 243.

[15] In this case, the district court determined that Dr. Debbie did *not* have actual knowledge of the specific danger in the March 29th experiment. Of course, it determined that Dr. Baer did not have actual knowledge of the specific danger either. Thus, the question becomes whether the knowledgeable user exception applies where the knowledge of both supplier and user is imputed, not actual. The government suggests that the exception should apply here, because the district court found that the state was more than twice as culpable as the federal government, and that Dr. Debbie's "failure to appreciate the hazard to which he was exposing his workers seems more unreasonable than Dr. Baer's failure to recognize the same hazard." *Andrulonis*, 724 F.Supp. at 1508.

[16, 17] While attractive, the government's argument is not dispositive, because the knowledgeable user exception applies only where the user is actually aware of the precise danger involved. *Belling*, 511 N.Y.S.2d at 733; *Kerr*, 557 F.Supp. at 287. A supplier's duty to warn, on the other hand, arises if he knows or should know of the danger; actual knowledge of the precise danger is not required to hold the supplier liable. *Schumacher*, 59 N.Y.2d at 248, 464 N.Y.S.2d at 441, 451 N.E.2d 195; *Young*, 383 N.Y.S.2d at 731. Here, the district court determined that the user had no actual knowledge of the precise danger, but both the supplier, Dr. Baer, and the user, Dr. Debbie, were charged with imput-

ed knowledge. In these circumstances, the government, through Dr. Baer's breach of his duty to warn, is liable to Andrulonis for his injuries.

Since we find that the duty-to-warn theory supplies a sufficient basis to establish the government's liability and to affirm that part of the district court's judgment, we need not address the district court's further reliance on the other two theories of liability.

C. *Allocation of Settlement Proceeds*

[18] Before the end of trial, the Andrulonises and the non-governmental defendants entered into a series of settlement agreements resulting in a total settlement of $1.3 million. The settling parties agreed to allocate $225,000 to Jerome Andrulonis and $1,075,000 to Joanna Andrulonis. Any settlement funds allocated to Joanna Andrulonis, whose claims against the government were dismissed for failure to timely file an administrative claim, could not be used to reduce the final award of damages to Jerome Andrulonis as provided under section 15–108 of New York's General Obligations Law. The government and NYSDOH now challenge this allocation and the district court's approval of it as being contrary to the policy underlying that section.

Section 15–108 of New York's General Obligations Law provides an incentive to settle and attempts to define the effect of a settlement on collateral rights and liabili-

ties in future litigation. Section 15-108(a) provides in relevant part:

> Effect of release of or covenant not to sue tortfeasors. When a release * * * is given to one of two or more persons liable or claimed to be liable in tort for the same injury, or the same wrongful death, it does not discharge any of the other tortfeasors from liability for the injury or wrongful death unless its terms expressly so provide, but it reduces the claim of the releasor against the other tortfeasors to the extent of any amount stipulated by the release or the covenant, or in the amount of the consideration paid for it, or in the amount of the released tortfeasor's equitable share of the damages under article fourteen of the civil practice law and rules, whichever is the greatest.

The "right of set-off" provided by subdivision (a) "is intended to avoid a result in which the non-settling tortfeasors bear more than their equitable share of the plaintiff's damages." *Lambert Houses Redevelopment Co. v. HRH Equity Corp.*, 117 A.D.2d 227, 232-33, 502 N.Y.S.2d 433, 436 (1st Dept.1986). The government and the state contend that the apportionment of settlement proceeds in the case at bar undermines this purpose. They claim that the Andrulonises intentionally allocated most of the settlement proceeds to Joanna Andrulonis in order to minimize any right

of setoff that might arise from trial of Jerome Andrulonis's remaining claims. Finally, they argue that the court should not have accepted the settling parties' decision, but should have independently allocated the settlement funds to reflect the relative injuries suffered by Jerome and Joanna Andrulonis.

In *Hill v. St. Clare's Hospital*, 67 N.Y.2d 72, 77, 499 N.Y.S.2d 904, 907, 490 N.E.2d 823 (1986), the injured party and his wife, as derivative claimant, entered into a settlement with the original tortfeasors, but continued their claim against subsequent tortfeasors for aggravation of the original injuries. Their general release contained no reservation of rights and no allocation of damages as between plaintiffs or as between the successive injuries. *Id.* Although the court focused primarily on the problem of allocating settlement proceeds between original injuries and aggravated injuries for purposes of setoff against the subsequent tortfeasors, its analysis is helpful in determining a proper apportionment between two plaintiffs.

The court stated that "[t]o the extent that the injured party and the original tortfeasor are permitted to stipulate the amount by which the liability of the successive tort-feasor is to be reduced by the original tort-feasor's payment, intent remains a factor, but the amount stipulated must also be shown to have been arrived at in good faith." *Id.* at 84–85, 499 N.Y.S.2d

at 912, 490 N.E.2d 823. The court determined that the finder of fact, in making a decision as to attribution of the settlement payment, should "consider the statement as to allocation, if any, * * * contained in the settlement documents and *the gravity of the respective injuries* and determine whether the amount allocated by the parties was arrived at in good faith." *Id.* at 86, 499 N.Y.S.2d at 912, 490 N.E.2d 823 (emphasis added).

In *Casey v. State*, 119 A.D.2d 363, 507 N.Y.S.2d 159 (2d Dept.1986), the plaintiffs sued three alleged tortfeasors for wrongful death and for conscious pain and suffering. Before trial, a settlement was reached with two of the defendants, but neither release separately allocated the proceeds to the wrongful death and the pain and suffering causes of action. Instead, the Surrogate's Court made the allocation decision. The court held that any settlement sum, even though allocated by the parties or by a decree of the Surrogate's Court, must, for the purpose of a subsequent setoff, be apportioned at the trial; and it is the obligation of the finder of fact, after hearing the evidence, to make such an apportionment. *Id.* 507 N.Y.S.2d at 162. A nonsettling party is not bound by any agreement as to allocation made without its knowledge or consent. *Id.* 507 N.Y.S.2d at 163. Therefore, the self-created apportionment proffered by the claimants and approved by the Surrogate was not binding on the trial court. *Id.*

In *Casey*, the court was concerned that the defendant state would be responsible for the bulk of a wrongful death award because the claimants' proposed allocation would have resulted in $175,000 of the settlement being allocated to a "potentially nonexistent pain and suffering cause of action", thereby unfairly reducing the setoff in the wrongful death action and leaving the state with none of the benefits of section 15–108. *Id.* Thus, the court, per Justice, now District Judge, Arthur Spatt, stated: "We are dealing with two separate and distinct causes of action. The State is entitled to a true setoff based on the merits of the respective causes of action. This apportionment [of the earlier settlement] must be predicated on the evidence adduced at the trial with regard to the monetary value of each cause of action." *Id.*

Similarly, in *Merrill v. State of New York*, 110 Misc.2d 260, 442 N.Y.S.2d 352 (1981), *aff'd*, 89 A.D.2d 802, 453 N.Y.S.2d 383, 384 (4th Dept.1982), where plaintiffs had earlier settled with one of the joint tortfeasors for a gross unallocated amount in a case involving separate claims for personal injuries and loss of services, the trial court held that the proper way to apportion the settlement proceeds was in proportion to the damages found to have been sustained by the respective claimants. *See also Hager v. Hutchins*, 91 Misc.2d 402, 398 N.Y.S.2d 316 (1977) (trial court confronted with a settlement with several defendants for an unallocated sum in a case

involving causes of action for wrongful death and conscious pain and suffering held that a prior order of the Supreme Court which had approved the compromise of the claims against the settling defendants and allocated the proceeds entirely to the wrongful death cause of action was not binding on the trial court).

The district court in this case assessed the reasonableness of the apportionment of the settlement monies and ultimately approved the allocation. It rejected the government's suggestion that *Hill* and *Casey* required that for setoff purposes, the apportionment of a settlement fund must in every instance approximate the relative injuries of the plaintiffs. Instead, the district court conducted a good faith analysis to determine whether the amounts allocated to Jerome Andrulonis and Joanna Andrulonis could be approved. First, it asked whether the amount set aside for Jerome Andrulonis was a reasonable approximation of the settling defendants' equitable share of his total damages, and second, whether the amount set aside for Joanna Andrulonis was unreasonably excessive in light of the damages she actually suffered.

The district court found that the amount allocated to Jerome Andrulonis did not grossly underestimate the settling parties' equitable share of the damages he suffered. Although Judge Munson noted a significant discrepancy between Lilly/Thompson's settlement amount and its

equitable share of Jerome Andrulonis's damages, he considered all the settling defendants as a group and determined that the total amount Jerome Andrulonis received did not so grossly deviate from the amount ultimately allocated to those defendants as to raise an inference of bad faith.

Judge Munson then turned to Joanna Andrulonis's portion of the settlement proceeds. In determining whether the total amount she received was a good faith assessment of the value of her claims, he applied the standard used to review a jury damage award—whether the award was so excessive as to "shock the judicial conscience". *See Martell v. Boardwalk Enterprises, Inc.*, 748 F.2d 740, 750 (2d Cir. 1984). The district judge felt this was the appropriate standard because a settlement precluded a right to a jury trial. Sensing no shock and looking to other damage awards in loss of consortium actions, the district judge concluded that the apportionment of settlement monies was made in good faith.

The district judge relied in part on the facts that Joanna Andrulonis was procedurally barred from any recovery against the government or the state, and that any amount she received from settling her claims with the private defendants represented the entirety of the damages she will recover. Thus, the nongovernmental defendants would bear the entire cost of the damages suffered by Joanna Andrulonis,

including that portion for which the United States and the NYSDOH would otherwise be responsible, but neither the government nor the state would be required to contribute.

We disagree with that analysis. Judge Munson approved the settling parties' allocation of settlement proceeds after determining whether it was "reasonable." New York law, however, requires the trial court to make an independent determination of what the proper apportionment of settlement proceeds should be, based on the monetary value of each cause of action. *See Casey*, 507 N.Y.S.2d at 163. Thus, the trial court should have made its allocation decision *de novo*, and not just reviewed the parties' chosen apportionment for reasonableness.

[19] In addition, while the fact that Joanna Andrulonis had no claim against either the government or the state may have influenced the parties in deciding whether or not to settle, it is not conclusive under New York law of the amount that can be allocated to Joanna Andrulonis upon a subsequent setoff. If Joanna Andrulonis had timely filed an administrative claim she also could have recovered damages from the government, and it may be unfortunate that she will not be able to recover from the governmental tortfeasors; but New York does not permit the parties' private agreements to decrease the amount of the settlement to be offset against the non-set-

tling defendants. The district court's point that only the settling defendants are paying for Joanna Andrulonis's injuries is incorrect, since the government and the state are, through the corresponding reduction in the setoff on Jerome Andrulonis's claim, being required to carry a significant portion of her award.

[20] Settling parties may not structure the apportionment to avoid a later setoff by a nonsettling defendant; to hold otherwise would permit them to circumvent the policy underlying section 15–108. The district court recognized that this is precisely what the Andrulonises had tried to do, but it felt that New York law did not prohibit such a result. We disagree. As shown above, the New York cases establish that the parties' settlement agreement, even when confirmed by a court order, does not bind a trial court in determining the proper allocation of settlement proceeds for purposes of setoff. That allocation must follow from a comparison by the trial court of the injuries of the settling plaintiffs. We therefore remand to the district court with a direction to independently evaluate the relative injuries of the settling plaintiffs and to reduce the damages to be awarded to Jerome Andrulonis by the amount of the settlement thus allocated to him.

D. *Apportionment of Culpability between the Government and NYSDOH*

[21] The district court, pursuant to N.Y.C.P.L.R. §§ 1401–1404 governing contribution claims, found the government responsible for 30 percent of the damages suffered by Jerome Andrulonis and NYSDOH responsible for 65 percent. NYSDOH appeals this apportionment and contends that the court erred in finding it responsible for more of the damages than the government. Relying on the notion that culpability is correlated with duty, which in this case happened to depend on the expertise of the actor, NYSDOH argues that the percentages of fault must be reassessed to reflect the federal government's greater responsibility in causing Jerome Andrulonis's illness. NYSDOH argues that the district court's finding that Dr. Baer was the preeminent expert in the field of rabies mandates that the government, having greater expertise, should be charged with a greater share of the damages.

We disagree. NYSDOH is correct in asserting that New York law holds experts to a higher standard of care. *See* Restatement (Second) of Torts § 289, comment m; *Toth v. Community Hospital at Glen Cove*, 22 N.Y.2d 255, 263, 292 N.Y.S.2d 440, 447–48, 239 N.E.2d 368 (1968) (holding specialists accountable to a higher standard

than general practitioners); *Hope v. Fall Brook Coal Co.*, 3 A.D. 70, 75, 38 N.Y.S. 1040, 1043 (4th Dept.1893) (duty of care is measured against knowledge and expertise of the participants). However, NYSDOH confuses application of the appropriate standard of care in defining the minimal level of duty that may be owed to another, with the entirely different process of weighing the expertise of an actor in determining his portion of damages. In essence, NYSDOH urges that the percentages of damages apportioned must be directly related to the relative levels of expertise of the defendants. New York law does not require such a restrictive approach.

[22] The district court's apportionment of damages, like other findings of fact, is subject to the clearly erroneous standard of review. Fed.R.Civ.P. 52(a). The district court relied on Dr. Baer's expertise in concluding that he had a duty to warn and, thus, in holding the government liable to Jerome Andrulonis. Turning to the degree of the state's culpability for purposes of contribution, the court had before it evidence of Dr. Debbie's expertise with the rabies virus, his prior experience with the Uni-Glatt machine, his intimate knowledge of conditions at the Griffin laboratory, and his role as supervisor of the laboratory with the attendant responsibilities. Balancing all the circumstances of the case, Judge Munson found Dr. Debbie's failure to appreciate the hazards of using the leaky machine in this series of rabies ex-

periments to be more culpable than Dr. Baer's single tragic lapse. Since his findings are not clearly erroneous, we affirm his apportionment of damages under N.Y. C.P.L.R. § 1402.

E. *Damages for Future Custodial Care*

[23] The district court awarded Jerome Andrulonis $2,417,238 for his future custodial care, relying on an assumption of continued care within the home. Jerome Andrulonis cross-appeals from this part of the judgment, claiming that the damage figure must be based on care in a neurological facility, since it is uncertain whether Joanna can continue to care for Jerome in her home indefinitely into the future. He asks that the damages for future custodial care be increased to $6,841,925.

This argument is without merit. The district court estimated future custodial care on the basis of 24-hour care with three shifts of nursing aides in his home for the rest of his life. Although professional nurses were not considered necessary for Jerome's daily care, the district court used the salary for professional nurses because no one else was willing to do the job at a lower cost. In addition, the district court also determined that Jerome would function best in the home environment where he could live in a predictable structure provided by his family. Since the plaintiff presented no evidence as to when, if ever, institutional care would replace the

completely supervised home care presently being provided for Jerome, the district court was not clearly erroneous in the amount it awarded for future custodial care.

CONCLUSION

We reverse that portion of the judgment of the district court that calculated the setoff under New York's General Obligations Law § 15–108 from the parties's own allocation of settlement proceeds and remand on that issue for further proceedings consistent with this opinion. In all other respects the judgment is affirmed.

Opinion of the United States District Court for the Northern District of New York, Dated November 15, 1989.

ANDRULONIS v. U.S.
Cite as 724 F.Supp. 1421 (N.D.N.Y. 1989)

Joanna ANDRULONIS, Individually, and as Conservator of the Property of Jerome Andrulonis, Plaintiffs,

v.

UNITED STATES of America; Glatt Air Techniques, Inc.; Glatt GmbH; Wisconsin Alumni Research Foundation, Inc.; WARF Institute, Inc.; Raltech Scientific Services, Inc.; Ralston Purina Company; Eli Lilly and Company; and John L. Thompson and Sons and Company, Defendants.

UNITED STATES of America, Third-Party Plaintiff,

v.

NEW YORK STATE DEPARTMENT OF HEALTH, Third-Party Defendant.

No. 79-CV-847.

United States District Court, N.D. New York.

Oct. 17, 1989.

As Amended Dec. 15, 1989.

Researcher who contracted rabies brought personal injury action, along with his wife, against New York State Department of Health, federal Government, and others. Following settlement with the other defendants, the District Court, Munson, J., held that: (1) employee of Center for Disease Control was negligent; (2) employee of New York State Department of Health who was directing the experiment was negligent; (3) negligence was proximate cause of injuries suffered; (4) failure of vaccine manufacturer to warn of possible ineffectiveness of vaccine against airborne transmission of rabies rendered product defective; (5) United States was 30% at fault, NYSDOH was 60% at fault, and drug manufacturer was 5% at fault; and (6) apportionment of settlement between claims of researcher and claims of his wife was not unreasonable.

Ordered accordingly.

Roemer and Featherstonhaugh, Albany, N.Y. (James D. Featherstonhaugh and John R. Mineaux, of counsel), for plaintiffs.

Dept. of Justice, Civ. Div., Torts Branch, Washington, D.C. (Leon Taranto and Anthony R. Sherr, Trial Attys., of counsel), and Frederick J. Scullin, Jr., U.S. Atty., N.D.N.Y., Albany, N.Y. (William Fanciullo, Asst. U.S. Atty., of counsel), for U.S.

Robert Abrams, Atty. Gen., State of N.Y., Albany, N.Y. (Kevan J. Acton and Robert Seigfried, Asst. Attys. Gen., of counsel), for defendant New York State Dept. of Health.

Anderson Russell Kill & Olick, P.C., New York City (R. Mark Keenan, of counsel), and Paul F. Donahue Associates, Albany, N.Y. (Alvin O. Sabo, of counsel), for defendant Glatt GmbH.

Carter, Conboy, Bardwell, Case and Blackmore, Albany, N.Y. (Philip J. Danaher, of counsel), for defendant Wisconsin Alumni Research Foundation.

McNamee, Lochner, Titus & Williams, Albany, N.Y. (Earl H. Gallup, Jr., of counsel), for defendants Raltech Scientific Services, Inc. and Ralston Purina Co.

Maynard, O'Connor & Smith, Schenectady, N.Y. (Richard Gershon, of counsel), for defendant-WARF Institute, Inc.

Ainsworth, Sullivan, Tracy and Knauf, Albany, N.Y. (Thomas F. Tracy, Frank J. Warner, Jr. and Margaret Comard Lynch, of counsel), for defendants Eli Lilly and Co. and John L. Thompson & Sons & Co.

Galef & Jacobs, New York City (Christopher M. Houlihan, of counsel), for defendant Glatt Air Techniques, Inc.

MEMORANDUM-DECISION AND ORDER

MUNSON, District Judge.

On March 29, 1977 the rabies virus invaded the body of plaintiff Jerome Andrulonis during an experiment conducted by his employer, the New York State Department of Health ("NYSDOH" or "the State"). The experiment was a part of the State's effort to develop a method for immunizing wildlife from the disease the virus causes. Shortly after being exposed to the virus, Andrulonis contracted the disease of rabies, and his central nervous system was ravaged. He survived the disease, thus becoming one of only three individuals in human history to do so, but the neurologic damage he suffered as a result of his illness left Andrulonis without the cognitive ability to appreciate this distinction.

On December 20, 1979 this action was commenced on behalf of Jerome Andrulonis and his wife, Joanna, against the United States of America ("United States" or "the Government") under the Federal Tort Claims Act ("FTCA" or "the Act"), 28 U.S.C. §§ 1346, 2671–2680, and against various non-governmental defendants, over whom jurisdiction was predicated on diversity of citizenship or the pendant party doctrine. An amended complaint adding additional non-governmental defendants was filed on October 9, 1981. Plaintiffs' claims sounded in negligence, strict products liability, and breach of warranty. The United States made cross-claims against the non-governmental defendants and filed a third-party complaint against NYSDOH, seeking contribution. *See* N.Y.C.P.L.R. §§ 1401–1404 (McKinney 1976). Subsequently, the court dismissed Joanna Andrulonis' derivative claims against the United States for failure to timely file an administrative claim, as required by the terms of the FTCA. *Andrulonis v. United States*, No. 79-CV-847, slip op. at 7 (N.D.N.Y. March 8, 1984); *see* 28 U.S.C. § 2675(a);

see also id. § 2401(b).[1] A trial on the remaining claims was conducted from February 18, 1987 until March 19, 1987. Before the end of the trial, plaintiffs entered into settlement agreements with all of the non-governmental parties,[2] leaving unresolved the FTCA claims of plaintiff Jerome Andrulonis against the United States and the contribution claims made by the Government. This memorandum-decision consti-

1. In addition, plaintiffs' claims against one of the non-governmental defendants, Dale Wurster, was dismissed for lack of in personam jurisdiction pursuant to Fed.R.Civ.P. 12(b)(2), *Andrulonis v. United States*, slip op. at 2 (N.D. N.Y. October 1, 1982), and the third-party claims of certain of the non-governmental defendants against NYSDOH employees John G. Debbie and Robert H. Huffaker were dismissed on eleventh amendment grounds. *Andrulonis v. United States*, slip op. at 5 (N.D.N.Y. September 24, 1984).

2. The terms of these settlement agreements are summarized *infra*, at note 655. Pursuant to these agreements, plaintiff Jerome Andrulonis, through his Conservator, Joanna Andrulonis, discontinued his claims against the non-governmental parties in exchange for payments totalling $225,000. Plaintiff Joanna Andrulonis discontinued her derivative claims against the non-governmental defendants for the consideration of $1,075,000. The United States has challenged the allocation of settlement monies for the purpose of determining its "right of set-off" under N.Y.Gen.Oblig.Law § 15-108 (McKinney 1978). This challenge is addressed in the text *infra*, at 1527-1528.

tutes the court's findings of fact and conclusions of law concerning the remaining claims.[3] *See* Fed.R.Civ.P. 52(a).

I. INTRODUCTION

In some ways, the case of Jerome Andrulonis is aberrational. Andrulonis is believed to be the only person to contract the disease of rabies who prior to any exposure to live rabies virus had developed through vaccination a significant level of rabies antibodies in his blood stream. He is one of only two individuals in this century known to be infected after being exposed to the rabies virus in a laboratory setting. As

3. The court's factual findings are presented in narrative form. While certain important credibility issues are expressly discussed in this memorandum-decision, for the most part the court does not attempt to address each and every conflict in testimony present in the record. Unless the context suggests otherwise, any citation to the testimony of a witness indicates that the court accepts that portion of that witness' testimony as credible and tacitly rejects contrary testimony. On a number of occasions during the trial the court reserved decision on objections to questions posed by counsel and allowed the witnesses to whom the questions were directed to answer. Most of the testimony elicited by the contested questions was not considered by the court, and the evidentiary objections that were made to testimony the court has disregarded will not be resolved. In making its findings in the course of this opinion, the court will address any unresolved objections to questions that elicited proof that was considered by the court.

will be seen, he is one of only four individuals in human history believed to have contracted the disease internasally, through what is referred to as the "aerosol route" or the "airborne route" of infection.[4] Nonetheless, upon examination of what was known about the transmission of the virus by the researchers involved with the experiment of March 29, 1977, what was considered safe and reasonable laboratory practice in the relevant scientific community at that time, and the manner in which the March 29, 1977 experiment was performed, it can only be concluded that Andrulonis' development of the rabies disease was the foreseeable and avoidable result of negligence on the part of the researchers involved with the project.

Because a lengthy narrative is to follow, a brief summary may give some context to the matters discussed. In March 1977 Je-

4. Trial Transcript ("Tr.") at 1031-32 (Testimony of Dr. Michael A.W. Hattwick); Tr. at 736 (Testimony of Dr. James R. Tillotson); United States Department of Health, Education, and Welfare, "Rabies in a Laboratory Worker—New York," 26 Morbidity and Mortality Weekly Report 183, 184 (1977) (admitted into evidence as Plaintiffs' Exhibit ("Exh.") 151 and hereinafter referred to as "Rabies in New York Laboratory Worker"); United States Department of Health, Education, and Welfare, "Follow-up on Rabies—New York," 26 Morbidity and Mortality Weekly Report 249, 249 (1977) (admitted into evidence as Exh. 79 and hereinafter referred to as "Follow-up on Rabies").

rome Andrulonis was a thirty-four year old senior bacteriologist employed by NYSDOH who was primarily involved with research conducted in the rabies laboratory at the State's Griffin Laboratory ("Griffin"), located near Albany, New York. Because he was constantly exposed to the rabies virus in his work, he had been immunized against the disease of rabies through the administration of a commercial vaccine manufactured by defendant Eli Lilly and Company ("Lilly") and distributed by defendant John L. Thompson and Sons and Company ("Thompson & Sons"). Periodically, Andrulonis received booster shots of the Lilly vaccine, which induced the creation of antibodies to the rabies virus in the blood stream ("serum antibodies"). The effectiveness of the Lilly vaccine was dependent on whether the rabies virus, once transmitted to an immunized individual, came into contact with serum antibodies before entering his central nervous system. In the typical case where the virus is transmitted to an individual by animal bite, the site of the bite wound is covered in the victim's blood, and the antibodies that had been created in the blood stream by the vaccine would have the opportunity to kill the invading virus. In the rare case where the virus is transmitted through the air and inhaled by the victim, however, the Lilly vaccine is probably ineffective. The olfactory nerves within an individual's nasal cavity are exposed to the air. If live rabies

virus comes into contact with an individual's olfactory nerve and infects it, the virus could travel the short distance from the olfactory nerve to the victim's brain without ever coming into significant contact with serum antibodies.

At the time Andrulonis was exposed to the virus that ultimately caused his illness, he was attempting to coat sugar nonpareils [5] with a solution containing live rabies virus. The coating process was accomplished through the use of a machine recommended by defendant WARF Institute, Inc. ("WARF").[6] The machine, known as the "Uni-Glatt," suspended the sugar nonpareils in a stream of upflowing air while various solutions were sprayed onto them. The Uni-Glatt Machine was manufactured by defendant Glatt GmbH and distributed in the United States by defendant Glatt Air Techniques, Inc., and employed an air suspension process that had been developed by Dale Wurster. The process created "aero-

5. Throughout the trial, the nonpareils were alternatively referred to as "pareils."

6. Originally, WARF was the wholly owned subsidiary of a Wisconsin corporation known as the Wisconsin Alumni Research Foundation ("Foundation"), a named defendant in this case.

Foundation sold its stock in WARF to defendant Ralston Purina Company in late 1979, and WARF changed its name to Raltech Scientific Services, Inc., also a named defendant in this case.

sols," which are suspensions of microscopic solid or liquid particles in air or gas. The Uni-Glatt machine was not airtight, and when the solution containing live rabies virus was sprayed onto the nonpareils, aerosolized virus escaped from the Uni-Glatt and into the atmosphere of the rabies laboratory at Griffin. The virus was inhaled by Andrulonis, apparently infected one of his olfactory nerves, and then multiplied and travelled from his olfactory nerve to his brain, causing severe and permanent damage.

The experiment in question was conducted under the supervision of Dr. John G. Debbie, a research scientist employed by NYSDOH. Also present during the experiment was Dr. George M. Baer, Chief of the Viral Zoonosis Branch and Rabies Laboratory at the Center for Disease Control ("CDC"), who conducted most of his work in the CDC's laboratories in Atlanta and Lawrenceville, Georgia. The CDC is a part of the United States Department of Health and Human Services, and in March 1977 was a subunit of the Department of Health, Education, and Welfare. The experiment was conducted in furtherance of a joint effort by scientists at NYSDOH and the CDC to develop a method for the mass immunization of wildlife from rabies. During the course of this effort, Debbie requested Baer to prepare a solution of highly concentrated rabies virus for experimental use in the Uni-Glatt machine. The virus "strain" Baer prepared at the CDC's

laboratory in Lawrenceville in response to this request was supplied to Debbie on March 28, 1977 and used in the Uni-Glatt machine the next day. At the time of the March 29, 1977 experiment, it was not known whether the virus Baer supplied was "pathogenic," that is, capable of causing disease in man. The court will find that during the course of the March 29 experiment, Andrulonis was exposed to an aerosol containing the virus Baer had prepared and as a result of this exposure contracted the disease of rabies.

Plaintiffs allege that the laboratory conditions under which the experiment was conducted at Griffin were unsafe, that Dr. Baer should have known this, and that Baer's failure to stop the experiment before Jerome Andrulonis was exposed to an aerosol containing rabies virus constituted negligence that was the direct and proximate cause of the injuries Andrulonis suffered. Plaintiffs also argue that Baer acted carelessly in supplying a highly concentrated virus strain to the comparatively inexperienced researchers at NYSDOH. It is asserted that Baer negligently failed to warn Dr. Debbie of the inherent dangers associated with the virus strain he provided and to which Andrulonis was exposed. Plaintiffs also maintain that the Government is liable because Dr. Baer and Dr. William G. Winkler, Chief of the Viral Zoonoses Section of the CDC's Viral Disease Division, negligently made representations

that induced WARF to recommend the use of the Uni-Glatt machine in experimental situations for which the machine was not suited. Finally, plaintiffs introduced evidence suggesting that the March 29, 1977 experiment was a part of a "joint venture" involving CDC and NYSDOH, and consequently that the United States is jointly and severally liable for any negligent acts on the part of NYSDOH employees, including Dr. Debbie. The Government denies any negligence on the part of its employees, and urges that the injuries plaintiff Jerome Andrulonis suffered were the result of his own negligence or the negligent acts of employees of the State or the non-governmental parties.

II. BACKGROUND

Unless the context indicates otherwise, this section of the opinion summarizes facts that reasonably should have been known as of March 29, 1977 by scientists who ventured beyond routine laboratory procedures involving the rabies virus and proceeded into the conception and supervision of experimental research projects involving that virus. Specifically, Drs. Debbie, Baer, and Winkler, whose conduct or representations are relevant to the claims made in this lawsuit, are charged with knowledge of the principles and theories discussed in the pages that follow, as well as familiarity with the research upon which those principles and theories were based.

In 1976 and 1977, these men were either members, or engaged in research projects that should only have been undertaken by members, of a select group referred to at trial as the "community of rabies experts," which in the United States numbered between twenty-five and fifty scientists.[7] Whether Jerome Andrulonis reasonably should have been aware of the body of

7. In 1976 and 1977, a small group of roughly twenty-five to fifty scientists were actively involved in serious research concerning the rabies virus in the United States. Tr. at 1251 (Testimony of Dr. H Fred Clark). Baer and Winkler were members of this core group. Tr. at 1251 (Clark); Tr. at 1397–98 (Testimony of Dr. Robert E. Shope). Beyond this group of rabies experts was a larger group of people who did diagnostic work or were more indirectly involved with the study of the rabies virus and the disease it causes. Tr. at 1251 (Clark). Some question as to Dr. Debbie's status in the rabies field in 1977 exists. Before 1970, Debbie was not involved in non-diagnostic rabies research. *See* Tr. at 97–98 (Testimony of Dr. John G. Debbie). Although he had a reputation as an excellent diagnostician, by 1977 Debbie's experience in rabies research was still comparatively limited. Tr. at 1412 (Shope). Notwithstanding this, Debbie testified that he was familiar with most of the studies discussed in the text. *See, e.g.*, Tr. at 255–59, 390–91, 414–15, 438 (Debbie). More importantly, much of the research he conducted at Griffin, including the experiments with the Uni-Glatt machine, was of the kind properly undertaken only by those who were members of the core group of rabies experts, and the court will charge him with the knowledge that reasonably should be possessed by members of that elite group. *See, e.g.*, Tr. at 1288–90 (Clark).

scientific knowledge discussed in this memorandum-decision is more problematical, and this issue will be discussed separately.[8] For the most part, the scientific literature that is cited by the court in this section predates the March 29, 1977 Uni-Glatt experiment, and the testimony cited concerns knowledge available to the relevant scientific community of rabies experts before that date.

A. *The Nature of the Rabies Virus*

Rabies is an acute infectious disease of the central nervous system that imperils a wide range of mammal species, including man. Historically rabies has been one of man's most dreaded diseases, no doubt because of its dramatic symptoms and the severe neurologic effects associated with it.

> The Greeks called rabies Lyssa or Lytta which meant madness. The disease in man was described as hydrophobia in which the sick person is tormented at the same time with thirst and the fear of water. The Latin word "rabies" comes from an old Sanskrit word *rabhas* which translated means "to do violence." The German word *tollwut* originates with the Indogermanic *Dhvar*, to damage, and

8. *See* text, *infra* at 1504–1508.

wut from middle German *wuot* which is rage. The French word for rage is derived from the noun *robere*, to be mad.[9]

As Jerome Andrulonis noted in his Master's Dissertation, "[e]ven today, the mere mention of this affliction is sufficient to strike terror into the hearts of the general public, for facts and folklore have blended to obscure any true understanding of this deadly malady." [10]

9. J. Steele, "History of Rabies," 1 *The Natural History of Rabies* at 1 (G. Baer, ed., 1975). *The Natural History of Rabies*, a two volume reference book, was admitted into evidence as Exh. 112 and will hereinafter be referred to as *Natural History*. This treatise was edited by Dr. George Baer of the CDC, one of the principals in the series of events leading to Andrulonis' contraction of the disease of rabies. The treatise, which is cited often in this opinion, was published before the events material to this lawsuit occurred. Dr. James H. Steele's chapter in *Natural History* will hereinafter be referred to as "History of Rabies."

10. J. Andrulonis, "ERA Rabies Grown in PK-15 Tissue Culture" at 1 (May 1973) (admitted into evidence as Government's Exhibit ("Exh.") G-121 and hereinafter referred to as "Andrulonis Dissertation").

The disease of rabies can develop in an animal after the introduction of the rabies virus into the body of that animal. The rabies virus is an "etiologic agent," in other words, it is an organism capable of causing disease. Infection by the rabies virus in nature usually occurs through the saliva of another infected animal during a bite. Although infrequent, the virus can also be transmitted through the milk of an infected animal or, in rare cases, internasally, if an animal is exposed to an aerosol containing the rabies virus.[11] Not every person or animal who comes into physical contact with an environment containing live rabies virus ("exposure" to the virus) has the live virus introduced into his or her body ("infection"), and not every person or animal infected with the rabies virus subsequently develops the disease of rabies.[12]

11. Tr. at 968 (Hattwick).

12. *See* M. Hattwick & M. Gregg, "The Disease in Man," 2 *Natural History* at 281–85 (hereinafter "The Disease in Man"). As used in this memorandum-decision, "disease" will refer to the development of clinical symptoms or sickness associated with the rabies virus' invasion of the central nervous system. *See id.* at 282. Those symptoms are summarized in the text, *infra* at 1437–1438.

A virus is a microscopic organism consisting of genetic material [13] surrounded by a protective protein shell. A virus can reproduce only within a living host cell.[14] The rabies virus is a neurotropic virus, or a virus that proliferates primarily in nervous tissue, although it also has an affinity for certain other tissues of a host that it has infected, including the tissue comprising the salivary and adrenal glands and the epithelial (surface) tissue of the lungs, bladder and urinary tract.[15] In the common case where the rabies virus is transmitted to a previously uninfected animal through the saliva of an infected animal during a bite, the rabies virus is introduced into the cells of the bitten animal's muscles, connective tissue, or nerves at the site of the bite.[16] Once introduced, the virus may fail to multiply, or may multiply within an infected cell at the location of the bite but not leave that cell, or may spread to

13. The genetic material is ribonucleic acid (RNA).

14. Tr. at 976 (Hattwick).

15. Exh. G-125C at 133 (Deposition of Charles V. Trimarchi).

16. G. Baer, "Pathogenesis to the Central Nervous System," 1 *Natural History* at 182 (hereinafter "Pathogenesis").

other cells locally but fail to invade the infected animal's central nervous system.[17] If any of these contingencies occur, sickness will not develop. On the other hand, if the virus reproduces at the site of infection, leaves that site and invades the victim's central nervous system, it will produce the disease of rabies in the infected animal.[18]

A common progression characterizes those cases where the disease of rabies develops in an animal or man after infection. At the site of exposure and infection, the virus goes through an incubation period lasting from ten days to several months,[19] and then invades nearby nervous tissue. The virus begins to travel along connected nerves until it reaches the central nervous system. The reproduction of the virus within the central nervous system can cause myelitis (inflammation of the spinal cord) or encephalitis (inflammation of the brain). As the animal becomes ill as a result of viral replication in the brain, the virus travels down nerve cells into the mouth and enters the diseased animal's saliva. Frequently, the infected animal will

17. "Pathogenesis," *supra* note 16, at 182.

18. "Pathogenesis," *supra* note 16, at 182.

19. "Pathogenesis," *supra* note 16, at 192; Tr. at 970 (Hattwick).

become aggressive or violent because of the encephalitic changes caused by the virus, and this effect will often coincide with the entry of the virus into the animal's saliva. The virus is transmitted to other animal hosts when an agitated rabid animal bites an uninfected animal, introducing saliva containing the deadly virus into the bitten animal.[20]

When rabies virus is introduced into the muscle tissue of an animal as a result of the bite of a rabid animal, the virus has entered by the intramuscular route of infection. The term "route of infection" refers to the manner in which the virus enters the body of an animal. For example, researchers can introduce the virus into a laboratory animal by the "parenteral route" if they inject the virus into the animal intramuscularly, intravenously, or subcutaneously (beneath the skin). In cases of infection by the parenteral route, researchers draw distinctions based on the proximity of the site of the parenteral invasion to the animal's central nervous system. In the laboratory, rabies virus can be introduced into an animal by the "intracerebral route," which is accomplished by injection of a solution containing virus directly into an animal's brain. Most importantly

20. "Pathogenesis," *supra* note 16, at 182-92; Tr. at 968 (Hattwick).

for the purposes of this lawsuit, rabies virus can infect an animal through the "airborne route" (alternatively called the "aerosol route") when the animal inhales aerosol suspensions containing rabies virus.

The chances that the disease of rabies will develop in an infected animal are closely related to the quantity of viral particles[21] to which the animal is exposed.[22] This is true regardless of the route of infection.[23] With respect to the aerosol route of exposure, the "dose" or quantity of aerosolized viral particles to which an animal is exposed is the most important factor in determining whether a rabies virus will likely cause disease in the animal as a result of this exposure.[24]

21. A viral particle, or virion, is a single, complete agent comprised of genetic material and a protein shell.

22. Tr. at 1856–57 (Testimony of Dr. William G. Winkler); Tr. at 1403, 1412, 1451–52 (Shope); *see also* Tr. at 1396–97 (Shope) (discussing aerosol exposures).

23. *See* Tr. at 1856–57 (Winkler) (indicating that rabies generally is a dose-related disease); Tr. at 1396–97 (Shope) (discussing aerosols).

24. *See* text, *infra* at 1447–1448.

As a species, man is not particularly susceptible to contracting the disease of rabies.[25] Nonetheless, between 1958 and 1972 there were an average of 709 reported cases of human rabies per year worldwide (virtually all of which resulted in death), and it is believed that this total significantly underestimates the number of human

25. "The Disease in Man," *supra* note 12, at 287–88. The likelihood that the disease of rabies will develop in a human being following an exposure to the rabies virus varies with the route of exposure and the quantity of virus to which the person is exposed. In cases where a man is exposed to the virus by the bite of an infected animal, the risk that disease will result is dependent on the species of the animal responsible for the human exposure and the location and severity of the bite. "The Disease in Man," *supra*, at 284. Even with the most serious exposures, the probability of developing the disease of rabies is less than one hundred percent. *Id.* at 287–88. The chances that an otherwise untreated human being will die as a result of a severe bite by a rabid dog to the fingers or hand has been estimated to be fifteen percent; the mortality rate when a rabid dog bites a human in the trunk or legs through torn clothing has been estimated to be three percent. *Id.* at 286; Tr. at 2149 (Testimony of Dr. George M. Baer).

cases worldwide.[26] The rabies virus is ordinarily transmitted to humans through the bites of wild and domestic animals. Exposure through rabid cats and dogs is still very prevalent throughout the world, although such exposure has been largely minimized in the United States in the past three decades as a result of effective animal control programs and strong policies favoring the vaccination of domestic animals. As a consequence of this development, the number of cases in which humans contracted the disease of rabies in this country decreased from an average of more than forty a year in the 1940s to an average of less than two a year in the 1960s.[27] The most recent cases of human rabies in this country for the most part have been attributable to the bites of bats, skunks, or raccoons.[28]

26. "The Disease in Man," *supra* note 12, at 287. A World Health Organization survey conducted for the year 1981 which accounted for under reporting estimated that 20,482 human deaths world-wide were attributable to the disease of rabies during that year, ninety-eight percent of those deaths occurring in Asia. *See* Tr. at 2274 (Baer).

27. "The Disease in Man," *supra* note 12, at 287.

28. Tr. at 969–70 (Hattwick).

The disease of rabies in man can be divided into five stages.[29] The first stage is the incubation period, which can last from ten days to several months, depending on the intensity of exposure and the quantity of virus to which the victim has been exposed.[30] The average incubation period is twenty to sixty days.[31] During this initial stage, the infected individual shows no signs of illness, and is usually perfectly well apart from symptoms related to the healing of any wound resulting from an animal bite.[32]

The incubation period ends when the victim experiences the first symptoms of sickness and the disease enters the prodrome stage. This stage usually lasts between one and five days, and the infected individual suffers from nonspecific flu-like symp-

29. *See* Tr. at 970–75 (Hattwick); Tr. at 696–97 (Tillotson).

30. Tr. at 970–71 (Hattwick). The intensity of an exposure is dependent in part on the duration of the exposure, the route of exposure, and the proximity of the exposure to the central nervous system. *See* Tr. at 1064 (Hattwick); "The Disease in Man," *supra* note 12, at 289.

31. "The Disease in Man," *supra* note 12, at 289.

32. "The Disease in Man," *supra* note 12, at 290; Tr. at 970 (Hattwick).

toms such as malaise, headache, fever, and fatigue. In cases involving animal bites, pain or numbness at the wound area is not uncommon. The victim may also exhibit anxiety, nervousness, agitation, or irritability.[33]

Shortly after the onset of prodromal symptoms, the disease will enter the acute neurologic stage and produce the unusual symptoms that are suggestive of the disease of rabies. At this point, the virus has entered the infected individual's brain and is multiplying. Intermittent hyperactivity and violence is common at this stage, and the victim typically suffers periods of agitation, thrashing, running, biting, screaming, or crying.[34] Hyperventilation, excessive salivation, and convulsions usually characterize this stage.[35] Between hyperactive episodes, the victim is lucid, though often anxious. Difficulties with automatic functions—breathing, swallowing, and salivating, for example—commonly arise. Indeed, attempts to drink water often will result in severe, painful spasms of the

33. "The Disease in Man," *supra* note 12, at 290; Tr. at 971 Hattwick); Tr. at 696 (Tillotson).

34. "The Disease in Man," *supra* note 12, at 290; Tr. at 971–72 (Hattwick).

35. The periods of hyperactivity are not dramatic in between five and twenty percent of reported cases of human rabies. Instead, paralytic symptoms predominate. "The Disease in Man," *supra* note 12, at 291.

pharynx and larynx, causing choking and gagging. The victim might develop hydrophobia, a psychic reaction to the sight of liquids causing spasms often resulting in strangulation. Historically, the fact that those who contract the disease of rabies develop great thirst but fear water was one of the most dreaded side-effects of the disease. Today this effect can be avoided by the performance of tracheostomies.[36]

If the victim does not die abruptly of suffocation or heart failure during the acute neurologic phase, paralysis gradually sets in. The victim becomes increasingly disoriented and confused, falls into a stupor, and ultimately slips into a coma.[37] The acute neurologic phase persists for between two and ten days, ending with the onset of coma. Historically, most victims of the disease have died at this stage, probably because of a cessation of breathing, although cardiac irregularities and spasms can occur.[38] If the victim survives, the coma phase can last for a few hours or for several months. At about this point in time, the virus is no longer damaging the victim, but a legion of potentially fatal complications can develop. Among those is an increase in intracranial pressure, cerebral

36. "The Disease in Man," *supra* note 12, at 290–91; Tr. at 971–73 (Hattwick).

37. "The Disease in Man," *supra* note 12, at 291.

38. Tr. at 974–75 (Hattwick); "The Disease in Man," *supra* note 12, at 291.

edema (an excessive accumulation of fluid in the brain substance), and internal hydrocephalus (an accumulation of cerebrospinal fluid in the brain).[39] Post-mortem studies of victims of the disease often reveal substantial damage to the brain matter caused by rabies encephalitis.[40]

The above-described neurologic effects of the rabies virus on an infected individual are so severe that the disease of rabies is almost always fatal. In all of human history, only three individuals have survived the coma stage to enter the fifth stage of the rabies disease, the recovery stage. One of those individuals is Jerome Andrulonis.

B. *Modern Rabies Research and the Efforts to Control the Disease in Man*

Although man's awareness of the disease of rabies dates to antiquity,[41] most scientific knowledge concerning the disease and the etiologic agent that causes it has been acquired within the last century. The roots of modern scientific research concerning

39. Tr. at 974–75 (Hattwick); "The Disease in Man," *supra* note 12, at 291, 292–94.

40. Tr. at 867–68 (Testimony of Dr. Lawrence D. Rodichok).

41. "History of Rabies," *supra* note 9, at 1.

rabies can be traced to the work of Louis Pasteur in the late nineteenth century.[42]

In Pasteur's time, dogs caused most human rabies deaths in the world, and thus Pasteur's initial work with rabies focused on the development of a vaccine for domestic dogs which would prevent the disease in that species by stimulating an active immune response to the agent that caused it.[43] Early in the course of his work, Pasteur observed that dogs which recovered

42. Before Pasteur's work in the 1880s, it had been demonstrated through crude and somewhat doubtful experiments that the disease of rabies could be transmitted by the saliva of an infected animal, and some investigators suspected that rabies was a disease of the nervous system. "History of Rabies," *supra* note 9, at 11–12. Scientists were unaware, however, that the causal agent was a living organism, much less how that organism caused the extreme physiological effects associated with the disease. *Id.* at 11. Pasteur theorized that the disease was caused by "a microbe of infinite smallness," L. Pasteur & M. Chamberland, "Nouvelle communication sur la rage," 98 C.R. Acad. Sci. (Paris) 457 (1884) (quoted in "History of Rabies," *supra* note 9, at 20), and he was the first investigator to demonstrate that "the central nervous system ... [is] particularly concerned and active in the development of the disease." "Pathogenesis," *supra* note 16, at 181 (quoting L. Pasteur, et al., 92 C.R. Acad. Sci. 1259 (Paris) (1881)).

43. *See* R. Sikes, "Canine and Feline Vaccines—Past and Present," 2 *Natural History* at 177 (hereinafter "Canine and Feline Vaccines").

from early symptoms of rabies after inoculation of liquid solutions ("suspensions") containing infected central nervous system tissue into the blood stream were immune to later inoculations. He also concluded that the injection of small amounts of "infective material"—it was not known that a virus caused rabies at the time—would not produce immunity. The problem was finding a way to inject "infective material" into an animal sufficient to yield an immune response without producing disease. Pasteur's solution was to "attenuate" the "infective material" before injecting it into the animal to be immunized.[44] "Attenuation" will be discussed in greater detail below.

By 1885, Pasteur had reported success in developing immune responses in experimental dogs after injecting them with a series of inoculations of suspensions of an attenuated rabies virus strain.[45] That same year, Pasteur relented to the pleas of

44. "History of Rabies," *supra* note 9, at 19–21.

45. "History of Rabies," *supra* note 9, at 20–21. The series of shots began with a non-pathogenic suspension of material taken from the spinal cords of rabbits infected with the rabies virus. The solution in each succeeding inoculation was progressively more pathogenic. H Clark, T. Wiktor, & H. Koprowski, "Human Vaccination Against Rabies," 2 *Natural History* at 341–43 (hereinafter "Human Vaccination").

the mother of a young boy who had been bitten by a rabid dog numerous times and administered a series of injections of the solution he had developed for dogs to the boy. The boy never developed rabies, and as a result of this apparent success Pasteur's work became so influential that it defined in large part the direction of modern rabies research well into the second half of the twentieth century. Many of the techniques used by Pasteur in his early work persisted in modern rabies investigation through 1976 and 1977, the time period in which the events most relevant to this lawsuit transpired, and discussion of these techniques will give context to terms used throughout the remainder of this opinion.[16]

1. The "Passaging" of Virus

In his efforts to develop a canine vaccine, Pasteur was the first rabies investigator to utilize a laboratory procedure known as "passaging" in order to adapt the etiologic agent that caused the disease of rabies (the rabies virus) to animal species other than the dog. After a certain number of "passages," a virus, through a process of natural selection, can become "virulent" (extremely pathogenic) for the host for which

46. *See* "Human Vaccination," *supra* note 45, at 342; "History of Rabies," *supra* note 9, at 19.

it is adapted. However, if a solution containing the passaged virus is injected into an animal with a cell structure different than that of the animal species for which the virus has been adapted, it is possible that the passaged virus will lack the capacity to cause disease in that animal.[47] Nonetheless, in some cases a suspension of passaged virus in an animal for which the virus has not been adapted will act as an antigen, a substance capable of inducing the animal's immune system to produce antibodies to the rabies virus.[48] An antibody is a protein with a structure such that it interacts only with either the antigen that induced its creation in the first place or an antigen closely related to the inducing antigen. If a passaged virus is capable of spurring the creation of antibodies, those antibodies once produced will be present to neutralize any rabies virus to which the immunized animal is subsequently exposed in the wild.[49]

47. *See* Tr. at 1256–57 (Clark); Tr. at 2155 (Baer); *Dorland's Illustrated Medical Dictionary* 138 (27th ed. 1988) (defining "attenuation").

48. *See* Tr. at 1798 (Winkler).

49. Exh. G-125G at 19–22 (Deposition of Dr. Franklin B. Peck); *Dorland's Illustrated Medical Dictionary* 88 (27th ed. 1988) (defining "antibody"); *see also* Tr. at 976 (Hattwick).

Basically, a "passage" is a defined period of growth for a virus through replication and mutation within a particular cell system. An animal, an embryonated egg, or a tissue culture is infected with a solution containing a given concentration of viral particles. The virus is allowed to incubate under certain conditions for a specified period of days, during which time the virus will multiply and mutate randomly. Presumably, viral particles well-adapted to the host animal or tissue culture will replicate more readily than viral particles that might be better adapted to other species or cell systems during the short-term evolution that takes place during the passage. A virus that multiplies more readily will also mutate more frequently. After a specified period of days has elapsed, live rabies virus will be "harvested" from the animal or tissue culture within which it was grown.[50] Under modern laboratory practice, passaging often transpires in tissue culture maintained in test tubes and bottles, and "harvesting" constitutes the removal of liquid containing live viral particles from the cells of the tissue culture. This liquid containing viral particles is called a "supernate."[51] Through passaging, rabies investigators seek to develop a "modified live virus ra-

50. Tr. at 1254–56 (Clark); Tr. at 2343–45 (Baer).

51. Tr. at 2343–44 (Baer).

bies strain" with certain predictable qualities. A "modified live virus" is any virus strain that has been altered through some laboratory process such as passaging and possesses certain constant, identified characteristics.[52]

In developing his vaccine, Pasteur isolated rabies "street" virus (the virus as it occurs in the wild) from the brain of a rabid cow in 1882. The street virus was injected intracerebrally into a rabbit, and after a certain amount of time had elapsed the virus was harvested. The harvested virus was then itself injected intracerebrally into another rabbit. This process was repeated until the virus had been passaged in rabbits ninety times.[53] The resulting virus strain

52. Tr. at 980–81 (Hattwick). Although different "strains" of rabies virus are distinguishable depending on the animal from which the virus is initially isolated or the animal or cell system through which the virus is subsequently passaged, *see* Tr. at 523–24 (Deposition of Dr. Melvin K. Abelseth), all strains cross-react in serum-neutralizing tests, discussed in the text, *infra* at 1442–1443. *See* H Clark & T. Wiktor, "Rabies Virus," in *Strains of Human Viruses* 177, 178 (Majer & Plotkin, eds. 1972) (admitted into evidence as one of a packet of documents constituting Exh. G–137 and hereinafter referred to as "Rabies Virus Strains"). Thus, the various rabies virus strains are generally considered of the same "serotype," inducing the same specific immune response in an animal infected by any of the various rabies virus strains. *Id.*

53. "Human Vaccination," *supra* note 45, at 343; "History of Rabies," *supra* note 9, at 20.

was more pathogenic to rabbits than street virus, and had a fairly constant incubation period of six to seven days when injected intracerebrally into rabbits. This virus strain proved to be less pathogenic than street virus for dogs, however, and it was repeatedly demonstrated in the laboratory that dogs inoculated with a series of injections of suspensions containing the virus strain not only did not develop disease but resisted disease after subsequent infection with a strain of rabies virus virulent to dogs (a so-called "challenge").[54]

The modified live virus strain Pasteur developed was a "fixed virus" with respect to rabbits but an "attenuated virus" with respect to dogs. A virus is deemed "fixed" for a particular species when it has a fairly specific incubation period—which is ordinarily comparatively short—and is always lethal to that species.[55] The characteristics

54. Tr. at 1257 (Clark); "Canine and Feline Vaccines," *supra* note 43, at 177; "Human Vaccination," *supra* note 45, at 343.

55. Tr. at 1796–97 (Winkler); Tr. at 882 (Deposition of Dr. William G. Winkler).

of a fixed virus are more predictable than those of a street virus.[56] A virus is "attenuated" for a certain species if it is less pathogenic for that species than street virus when introduced into an animal by a specified route.[57] Almost invariably, when scientists attempt to attenuate a live virus for a particular species, it is done in an attempt to develop a vaccine against rabies for that species.[58]

A rabies vaccine is a suspension of either attenuated live rabies viral particles (a "modified live rabies virus vaccine") or inactivated rabies viral particles (a "killed virus vaccine") which is administered to an animal host in order to stimulate an immune response in that host without induc-

56. Tr. at 1797 (Winkler). The two most common examples of fixed viruses used by modern rabies researchers are the PV virus and the Challenge Virus Standard (CVS virus). The PV virus is derived from the original virus strain developed by Pasteur. It is maintained by the Division of Biologic Standards of the National Institute of Health and is used for vaccine production. The CVS virus is derived from the original Pasteur strain by further passage in mouse brain. It is the uniform challenge virus used in rabies investigations. *See* Tr. at 1799 (Winkler).

57. Tr. at 1798 (Winkler); Tr. at 1253 (Clark); Tr. at 978–90 (Hattwick); Tr. at 634 (Abelseth Deposition).

58. Tr. at 1798 (Winkler); Tr. at 978–79 (Hattwick).

ing disease.[59] Strictly speaking, the term "vaccine" is properly used to describe a specific suspension that has been licensed for use as a vaccine for a particular species.[60] It is important to note that a virus strain, particularly an attenuated live virus strain, that has been tested and appr ed for use as a vaccine for one animal species may be pathogenic if introduced into another species.[61] Moreover, a virus strain that has been shown to be a safe and effective vaccine when introduced into an animal by a specified route may prove to be pathogenic when introduced into the same animal by a different route.[62] This is not surprising given the process by which a live virus is attenuated in order to make a vaccine: to make the virus non-pathogenic for one species, the virus is made to thrive in a cell culture different than that of the species for which the vaccine was developed.[63] As a general rule, only by conducting labo-

59. *See* Tr. at 976, 980–81 (Hattwick); Tr. at 2041 (Richardson); *Dorland's Illustrated Medical Dictionary* 1431 (27th ed. 1988) (defining "vaccine").

60. Tr. at 981 (Hattwick).

61. Tr. at 979–80 (Hattwick).

62. Tr. at 979–80 (Hattwick); Tr. at 1256 (Clark).

63. Tr. at 1256 (Clark).

ratory tests in which a particular modified live virus strain is introduced into a particular species by a specified route can it be reliably determined whether that virus strain is attenuated for the species in question when infected by the virus through the route specified.[64]

2. Control of Human Rabies in the United States: The Mass Immunization of Canines and the Lilly Vaccine

The immunization method developed by Pasteur required multiple inoculations, and was never adopted for the mass immunization of dogs. It was not until the early 1920s that a practical one-dose vaccine for dogs was developed in Japan.[65] The virus strain used in the vaccine had been fixed for rabbits and then inactivated,[66] a process by which the biological activity of the virus is destroyed either through exposure to heat, chemicals, or ultraviolet irradiation.[67]

64. *See* Tr. at 1854 (Winkler).

65. E. Tierkel, "Control of Urban Rabies," 2 *Natural History* at 190 (hereinafter "Urban Rabies"); "Canine and Feline Vaccines," *supra* note 43, at 177–78.

66. "Canine and Feline Vaccines," *supra* note 43, at 178.

67. *See* "Human Vaccination," *supra* note 45, at 345–47 (discussing some of the methods that have been used to either partially or completely inactivate rabies virus).

Although this vaccine proved effective in controlling rabies in dogs in Japan and was used in other countries, use of the vaccine in the United States was sporadic.[68] In the late 1920s, it was discovered that some lots of that vaccine contained live virus, and thereafter different methods of inactivation were tested. In the course of these tests, it was found that inactivation rendered many lots of vaccine ineffective in stimulating an immune response in animals. Since there was no reliable way to determine the antigenic efficacy of a particular vaccine lot before it was used, efforts to implement an effective mass immunization programs were frustrated.[69]

The obstacle to mass immunization posed by the inability to pre-determine vaccine efficacy was overcome in the 1940s with the development of reliable standardized tests for both the evaluation of vaccine potency and an animal's immune response to the administration of a vaccine.[70] These tests, known as "titrations," are used to yield a standardized number called a "titer." In essence, "titer" is a term that is

68. "Canine and Feline Vaccines," *supra* note 43, at 178; "Urban Rabies," *supra* note 65, at 190.

69. "Canine and Feline Vaccines," *supra* note 43, at 179.

70. *See* Canine and Feline Vaccines," *supra* note 43, at 179; "Urban Rabies," *supra* note 65, at 190.

used to indicate a measure of a substance's ability to cause some defined result. By assigning a titer to a particular solution containing a virus, for example, researchers can compare that virus solution to other solutions containing the same virus with respect to the ability to cause the result contemplated.

When rabies researchers measure the "titer" of a rabies virus strain, they are measuring the ability of that virus strain to kill laboratory mice after intracerebral injection of a solution containing that virus strain.[71] The titer of a rabies virus strain is measured in units of "50% mouse intracerebral lethal doses per .03 milliliters (MICLD-50/.03 ml)," a reference to the number of dilutions of the virus strain that could be injected intracerebrally into a group of mice in .03 milliliter suspensions and induce disease in fifty percent of the mice injected.[72] A virus strain with a high

71. Tr. at 547 (Abelseth Deposition); Tr. at 408 (Debbie). Laboratory mice have been chosen as the standard species for titration because of their acute susceptibility to neurotropic viruses such as the rabies virus and their ready availability to research laboratories. J. Thomas, "The Serum Neutralization, Indirect Fluorescent Antibody, and Rapid Fluorescent Focus Inhibition Tests," 1 *Natural History* at 420 (hereinafter "Serologic Tests"); "History of Rabies," *supra* note 9, at 26.

72. *See* Tr. at 317-18 (Debbie).

titer has a greater capacity to kill mice than a virus strain with a low titer.[73] The titer is commonly expressed exponentially, with a base number of ten raised to a certain power. For example, a virus strain could have a titer of ten raised to the power of 3.5 MICLD–50/.03 ml. The "common logarithm" of the titer represented by the foregoing mathematical expression is 3.5. For the remainder of this memorandum-decision, the titer of a virus strain will be expressed by reference to its common logarithm.

Rabies investigators also measure an animal's immune response to a vaccine by reference to the term "titer." Here, "titer" is a measurement of neutralizing antibodies in an immunized animal's blood stream.[74] Blood serum isolated from a blood sample of the immunized animal is added to a solution containing the challenge virus standard ("CVS"), a fixed standardized rabies virus strain with constant characteristics derived from the original Pasteur virus. This mixture is incubated and then inoculated into mice. The measure of antibodies in the blood serum is determined by evaluating their ability to

73. Tr. at 547 (Abelseth Deposition).

74. Tr. at 977 (Hattwick); *see* "Serologic Tests," *supra* note 71, at 420–23.

neutralize the viral particles present in the CVS strain and prevent mouse deaths. Serum neutralizing antibody titer is usually expressed as a ratio, such as 1:5. The higher the second number in the ratio, the greater the amount of viral particles that can be neutralized by the immunized animal's serum antibodies.[75] Hereafter in this opinion, references to "titer" allude to the measure of the ability of a rabies virus strain to kill laboratory mice, while the phrase "antibody titer" will be used to refer to the level of neutralizing antibodies in an animal's blood stream.

As a general rule, if the titer of a particular virus strain is increased by some laboratory procedure, the pathogenicity of that virus strain is increased for species for which it has not been attenuated.[76] It appears that the most important factor determining the magnitude of the titer of a suspension of a modified live virus strain is the quantity and concentration of viral par-

75. "Serologic Tests," *supra* note 71, at 422–23.

76. Tr. at 1126 (Deposition of Dr. George M. Baer). This is not an absolute rule; the pathogenicity of a virus strain can vary from species to species, and "titer" measures the relative pathogenicity of a virus strain only with respect to mice. Tr. at 408 (Debbie); Tr. at 587–88 (Abelseth Deposition).

ticles in the suspension.[77] The titer of a particular virus strain can be increased either by passaging or by concentration.[78] By passaging a virus strain in animals, embryonated eggs, or cell cultures, the virus replicates within the vehicle chosen for passaging, thus increasing the quantity of virus obtained.[79] By concentrating a virus strain through one of a variety of available laboratory techniques, live viral particles are condensed into a smaller volume of

77. *See* Tr. at 1856–57 (Winkler) (indicating that the titer of a virus strain is directly related to the "dose" or quantity of viral particles contained within a given volume of virus). The measure of a virus strain's titer, however, is not completely dependent on the concentration of the viral particles it contains. Titer is a measurement of a virus strain's pathogenicity in mice, *see* Tr. at 408 (Debbie), and a high titer measurement of a virus strain conceivably could indicate that that strain is particularly well adapted to mice, and not that it is more concentrated than a lower titer virus.

78. *See generally* Exh. G–125C at 379–83 (Trimarchi Deposition); T. Wiktor & H Clark, "Growth of Rabies Virus in Cell Culture," 1 *Natural History* at 155–77 (hereinafter "Growth in Cell Culture"); L. Schneider, "Concentration and Purification," 1 *Natural History* at 125–38 (hereinafter "Concentration and Purification").

79. Tr. at 1811 (Winkler); *see* "Growth in Cell Culture," *supra* note 78, at 171–73.

virus solution.[80] Passaging a virus strain results in a changed virus strain that can be more pathogenic for a particular species either because of the mutations that the virus underwent during passaging, or because of an increase in the number of viral particles in a given volume of a suspension of the virus strain.[81] Concentration of a virus strain will not result in viral replications and mutations,[82] but does increase the number of viral particles in a given volume of a suspension. Because the pathogenicity of a rabies virus exposure is dose-related, concentration can also increase the pathogenicity of a virus strain.[83]

After the development of a reliable and convenient means of pre-testing the antigenic efficacy of a particular vaccine lot, a demonstrably effective mass canine immunization program was carried out in the United States for the first time. The program, instituted in Memphis, Tennessee in 1948, combined mass vaccination of dogs and strict animal control measures.[84] In

80. Tr. at 1413 (Shope).

81. Tr. at 1255–57 (Clark); *see also* Tr. at 1793–94 (Winkler).

82. *See* Tr. at 2115–16 (Baer).

83. Tr. at 1856–57 (Winkler); Tr. at 1413 (Shope).

84. "Urban Rabies," *supra* note 65, at 191.

the two decades that followed, similar programs were instituted throughout the United States, severely limiting the incidence of rabies in domestic dogs.[85] With the control of rabies in domestic dogs, reports of the disease in humans in this country dropped to near zero.[86]

Just as the control of rabies in dogs markedly reduced the incidence of human exposure to the rabies virus in the United States, significant improvements were made in the vaccines available for the treatment of those humans who were exposed to the virus. Until the 1950s, the vaccines developed for human use were variations of the Pasteur vaccine that originally had been intended for use in dogs and later adapted for human immunization.[87] The Pasteur vaccine had been developed through viral passages in the brains of living rabbits, and the modified live rabies virus strain that resulted was suspended in

85. "Urban Rabies," *supra* note 65, at 193–96.

86. "History of Rabies," *supra* note 9, at 27–28; Tr. at 2137 (Baer).

87. "Human Vaccination," *supra* note 45, at 343–47.

solutions of the spinal cords of rabbits.[88] The administration of any vaccine containing adult animal nervous tissue presents a risk of inducing allergic reactions resulting in neuroparalytic disorders.[89] In an effort to avoid this serious side-effect, defendant Lilly developed a vaccine in the 1950s that did not contain the nervous tissue of adult animals.[90]

The duck embryo vaccine ("DEV" or "the Lilly vaccine") was produced by passaging the CVS fixed virus strain in seven-day-old embryonated duck eggs, a process which yielded a high-titer supernate upon harvest.[91] The virus was inactivated and prepared for use as a vaccine. Similar to the original Pasteur vaccine, DEV was to be administered in a series of fourteen to twenty-one inoculations, and when it was licensed and first distributed in 1957, it was intended exclusively for post-exposure

88. "Human Vaccination," *supra* note 45, at 343. Live rabies virus was contained in the vaccine Pasteur originally used in a human. Scientists subsequently utilized methods for the partial or complete inactivation of the virus in vaccines used for human immunization. *Id.* at 346–47.

89. "Human Vaccination," *supra* note 45, at 347, 356–60; "History of Rabies," *supra* note 9, at 25.

90. "Human Vaccination," *supra* note 45, at 348.

91. Tr. at 565–66 (Abelseth Deposition); Exh. G–125G at 172 (Peck Deposition); "Human Vaccination," *supra* note 45, at 348.

treatment.[92] In the mid–1960s, defendant Lilly amended the package insert that was routinely distributed with the lots of the vaccine it manufactured and recommended it for pre-exposure use for "high-risk" individuals.[93] Among those considered "high-risk" were veterinarians, spelunkers, and laboratory personnel working with rabies virus.[94] It was common practice by the early 1970s for scientists and laboratory technicians engaging in rabies research to maintain a minimum level of serum antibodies through periodic inoculations with the Lilly vaccine.

92. "Human Vaccination," *supra* note 45, at 348; Exh. G–125G at 25, 89 (Peck Deposition). Post-exposure treatment with the Lilly vaccine was considered feasible because the rabies virus generally has a fairly long incubation period when introduced parenterally. The theory was that serum antibodies could be created by intensive post-exposure inoculations with DEV and neutralize the rabies virus already present before that virus entered the victim's central nervous system, causing disease. Exh. G–125G at 21–25 (Peck Deposition); "Human Vaccination," *supra*, at 341.

93. Exh. G–125G at 53–54 (Peck Deposition); *see also* Exh. 87A (Package insert supplied by Lilly for Rabies Vaccine, USP (Duck Embryo) Dried Killed Virus for the Prevention of Rabies (as revised November 14, 1975)).

94. Exh. 87A (Lilly package insert).

Although other non-nervous tissue vaccines were available for use in man, DEV was the vaccine of choice for human use in the United States from the late 1950s until 1980.[95] When the Lilly vaccine was being tested for safety and effectiveness before it was licensed, defendant Lilly focused on whether DEV would provide protection against disease in the event of human exposure to the rabies virus through a bite or scratch wound or a pre-existing abrasion after contact with the saliva of an infected animal (hereinafter collectively referred to as the "bite route" of exposure).[96] At the time, the bite route was the only known means of transmitting the virus in nature,[97] and thus the protectiveness of the Lilly vaccine against development of the disease of rabies after infection through a non-bite route was not investigated.[98] Specifically, no tests concerning the effectiveness of DEV against aerosol exposures to the rabies virus were conducted.[99]

95. Tr. at 1057 (Hattwick); Tr. at 419 (Debbie).

96. Exh. G-125G at 24–25 (Peck Deposition).

97. *See* W. Winkler, "Airborne Rabies," 2 *Natural History* at 115–17 (hereinafter "Airborne Rabies"); Tr. at 1237–38 (Clark).

98. Exh. G-125G at 25 (Peck Deposition).

99. Exh. G-125G at 31 (Peck Deposition).

C. *The Airborne Transmission of the Rabies Virus*

The belief that the rabies virus was transmitted in nature only through the introduction of the saliva of an infected animal into a bite wound or open lesion of another animal persisted until the late 1950s or early 1960s.[100] This opinion had endured in the scientific community despite scattered reports in the early medical literature indicating that animals could acquire the disease of rabies by inhaling the virus.[101] In the late 1950s, scientists began

100. "Airborne Rabies," *supra* note 97, at 115–17; Tr. at 1237–38 (Clark).

101. In a leading early treatise on the disease of rabies, it was reported that two humans who had not been bitten or scratched died from the disease after feeling the "hot breath" of a rabid wolf on their faces. "Airborne Rabies," *supra* note 97, at 115 (citing V. Babes, *Traité de la rage* (1912)). Possibly because these cases predated modern research methods, little significance was attached to them. In 1938, two researchers exposed laboratory rodents to mechanically generated "mists" containing rabies virus particles, and some of the rodents were infected. "Airborne Rabies," *supra*, at 115 (citing P. Remlinger & J. Bailly, 24 Bull.Acad.Nat.Med. (Paris) 72–74 (1938)). In 1940, Dr. Albert Sabin, an American scientist, inoculated laboratory mice intranasally with a fixed rabies virus with a titer of approximately 6 or 7, causing infection in practically all of the mice so exposed. Sabin found in those experiments that the virus initially invaded the olfactory regions of the brain and then disseminated throughout the rest of the brain. Tr. at 1237–38 (Clark). Apparently, it was not believed that the results obtained in the laboratory in these two sets of experiments revealed anything about the transmission of the rabies virus in the wild.

to question the assumption that direct contact with the saliva of an infected animal was necessary in order for the rabies virus to be transmitted to another animal.

1. The Discovery that Animals Can be Infected after Exposure to Aerosols Containing Rabies Virus

In 1956, a thirty-eight year old public health worker contracted the disease of rabies soon after being exposed to the atmosphere of various bat caves in the southwestern region of the United States. One of the caves this worker had entered was the Frio Cave in Uvalde County, Texas, which was known to shelter rabid bats. While the public health worker had worked with the rabies virus on occasion in a laboratory setting, there was no clear evidence that he had been exposed to the virus either by animal bite or laboratory accident.[102] In 1959, a mining engineer who was examining guano deposits in various bat caves in the Southwest developed the disease. Before his death, the engineer

102. Tr. at 1238 (Clark); "Airborne Rabies," *supra* note 97, at 116. The worker handled a number of bats in Frio Cave while wearing gloves, and he had an area of chronic dermatitis on his neck. Thus, it was possible that the rabies virus invaded through a lesion on the worker's neck. D. Constantine, "Rabies Transmission by Air in Bat Caves," Pub. Health Serv. Publ. No. 1617 (CDC) at 1 (June 1967) (admitted into evidence as Exh. 152 and hereinafter referred to as the "Constantine Report").

insisted that he had not been bitten or scratched by a bat or other animal. This individual had also explored Frio Cave, and thus was exposed to the atmosphere of a cave housing rabid bats.[103] The deaths of these two men suggested that it was possible that an unknown method of rabies virus transmission existed.[104] A number of field studies and laboratory experiments followed. The most important of these was a series of experiments conducted on behalf of the CDC under the direction of Dr. Denny G. Constantine in which various mammals were exposed to the atmosphere of Frio Cave.[105]

Frio Cave is a large multi-chambered cavern which provides shelter for three species of bats. Most numerous are the Mexican free-tailed bats, whose numbers varied sea-

103. Tr. at 1238 (Clark); "Airborne Rabies," *supra* note 97, at 116. A co-worker reported that the engineer had a small bleeding scratch wound when he emerged from Frio Cave. *See* G. Humphrey, G. Kemp, & E. Wood, "A Fatal Case of Rabies in a Woman Bitten by an Insectivorous Bat," 75 Pub. Health Rep. 317, 324 (1960) (admitted into evidence as one of a packet of documents constituting Exh. G–137 and hereinafter referred to as "Insectivorous Bat").

104. Constantine Report, *supra* note 102, at 1.

105. Tr. at 1239 (Clark); "Airborne Rabies," *supra* note 97, at 116.

sonally from between two and upwards of twenty million.[106] Millions of bats, primarily lactating females and their suckling, flightless young, were present in just one chamber of the cave (the "nursery chamber") from June until early August. Most of Constantine's work was conducted in this chamber.[107] The bats in the nursery chamber clung to a ceiling that ranged from four to ten feet in height. The concentration of bats in the nursery chamber varied from three hundred to four hundred per square foot.[108] The ventilation in the chamber was poor, and a continual rain of saliva, urine, and feces fell from the bats to the ground.[109]

In July 1960, thirteen carnivores confined in mesh cages were placed in the nursery chamber, where they remained for a period of seven days. A number of rodents were placed in the same chamber for lesser periods of time. While none of the rodents contracted rabies, four of the carnivores

106. Constantine Report, *supra* note 102, at 1.

107. *See* W. Winkler, "Airborne Rabies Virus Isolation," 4 Bull. Wildlife Disease Assoc. 37, 37 (1968) (admitted into evidence as one of a packet of documents constituting Exh. G-137 and hereinafter referred to as "Airborne Isolation").

108. Tr. at 2247–48 (Baer).

109. Tr. at 2249 (Baer); *see also* "Airborne Rabies," *supra* note 97, at 117.

were killed by the disease.[110] Although Constantine could not rule out the possibility that the animals were bitten through their mesh cages by diseased bats or other animals present in the nursery,[111] the suggestion that the caged animals contracted rabies as a result of exposure to rabies viral particles suspended in the atmosphere of Frio Cave was strong.[112]

In experiments conducted in Frio Cave in July 1961, elaborate steps were taken to assure that some of the animals exposed to the atmosphere of the nursery chamber would not be subject to other exposures from animal bites or arthropods.[113] These animals—four coyotes and four grey foxes—were confined in the chamber between

110. Constantine Report, *supra* note 102, at 12, 18, 42.

111. Constantine Report, *supra* note 102, at 12; Tr. at 1239–40 (Clark).

112. Dr. William G. Winkler went so far as to state that "[t]he transmission of rabies virus by non-bite route was demonstrated" by the July 1960 experiment. "Airborne Isolation," *supra* note 107, at 37.

113. For example, the animals were confined in sealed cages with especially fine, reinforced mesh at either end to allow air to filter through, and moats of glycerine-soaked spun glass padding were constructed just inside the exposed mesh at each end of the cages to prevent tiny crawling arthropods from invading the cages. Constantine Report, *supra* note 102, at 13.

twenty-four and thirty days. All subsequently died of rabies.[114] From these and other experiments conducted at Frio, Constantine concluded that it was "apparent that transmission of rabies virus from freetailed bats to other animals occurs by air." [115]

Constantine's experiments were conducted in poorly ventilated caves that contained an unusually large number of bats.[116] Any intimation that the airborne transmission of the rabies virus was limited to the unique environment of Frio Cave, however, was quickly dispelled. In 1967, a spontaneous outbreak of rabies occurred in the laboratory animal colony of the United States Public Health Services's Southwest Rabies Investigation Station at Las Cruces, New Mexico. Caged foxes, coyotes, and opossums which were never exposed to direct physical contact with a rabid animal began dying of rabies. Over a little less than ten months, sixty-four animals died, and thirty-nine of those animals had never been intentionally exposed to the rabies virus by researchers.[117] After an extensive investiga-

114. Constantine Report, *supra* note 102, at 18.

115. Constantine Report, *supra* note 102, at 26.

116. "Airborne Rabies," *supra* note 97, at 117.

117. W. Winkler, E. Baker, & C. Hopkins, "An Outbreak of Non-Bite Transmitted Rabies in a Laboratory Animal Colony," 95 Amer. J. Epidem. 267, 267–68 (1972) (admitted into evidence as one of a packet of documents constituting Exh. G-137 and hereinafter referred to as "Non-Bite Transmitted Rabies"); "Airborne Rabies," *supra* note 97, at 118; Tr. at 1244 (Clark).

tion led by Dr. William G. Winkler of the CDC, it was concluded that the outbreak was most likely attributable to the airborne transmission of the rabies virus.[118] Animals that had been exposed to rabies virus in experiments were moved in and out of a room where animals that had not been exposed were housed, and infective virus was probably circulating in that room during the period of the outbreak. It was believed that the aerosols were generated either by the exposed animals themselves or as a result of a cage cleaning procedure that utilized a high-pressure spray.[119]

In 1973, the death of a fifty-six year old veterinarian underscored the potential hazard aerosols containing rabies virus posed to laboratory workers. The victim had no known animal bite exposures prior to falling ill, and no lesions on his body were observed during an autopsy performed after his death.[120] The veterinarian was an employee of a commercial laboratory in

118. "Non-Bite Transmitted Rabies," *supra* note 117, at 276.

119. "Non-Bite Transmitted Rabies," *supra* note 117, at 275.

120. W. Winkler, et al., "Airborne Rabies Transmission in a Laboratory Worker," 226 J.Amer. Med.Assn. 1219, 1220 (1973) (admitted into evidence as one of a packet of documents constituting Exh. G-137 and hereinafter referred to as "Laboratory Worker").

Texas that was involved in the preparation of various anti-viral vaccines. In preparing an experimental lot of animal rabies vaccine, he used a common "kitchen-type" blender to homogenize goat brains that had been infected with the CVS rabies virus strain. The blender produced aerosols, and it was hypothesized that the veterinarian "inhaled substantial amounts of virus" while transferring the homogenized goat brains from the blender to other containers.[121] The liquid homogenate in the blender had a titer of 6.5.[122] The veterinarian had had no other known contact with the rabies virus for a number of years before his death, and he succumbed to the disease of rabies shortly after this laboratory incident.[123] The veterinarian, who apparently was not effectively immunized against rabies,[124] was the first individual suspected to

121. "Laboratory Worker," *supra* note 120, at 1219–20; Tr. at 1245 (Clark).

122. Tr. at 1005 (Hattwick); "Airborne Rabies," *supra* note 97, at 119.

123. Tr. at 1245 (Clark).

124. The veterinarian had been immunized against the rabies virus thirteen years before his death, but had not received booster shots after the initial immunization. "Laboratory Worker," *supra* note 120, at 1220.

have died as a result of an aerosol exposure to a fixed strain of virus rather than a wild or "street" strain.[125]

2. The Pathogenesis of Airborne Rabies Infection

The death of the Texas veterinarian provided dramatic evidence that the newly-discovered route of infection could have important ramifications for laboratory workers using rabies virus strains. Even before this incident, however, there was some evidence suggesting that aerosols containing rabies virus could pose a threat to man different in kind than that presented by the more common exposures through animal bites or accidental injection in the laboratory. Constantine advised that "the term 'susceptibility' in reference to transmission by air route may constitute a marked departure from the term in its usual connotation regarding rabies."[126] At the time Constantine prepared his report summarizing his studies in Frio Cave, "[n]o attempt [had] been made to determine the actual virus invasion sites" in the two men who were suspected to have died as a result of their exposure to the rabies virus in Frio Cave in the 1950s.[127] It was recognized that a proper assessment of the dan-

125. "Laboratory Worker," *supra* note 120, at 1219; Tr. at 1246 (Clark).

126. Constantine Report, *supra* note 102, at 26.

127. Constantine Report, *supra* note 102, at 28.

ger which aerosols containing rabies virus posed to man and other animals would require study of the means by which the virus travelled through the body of an animal from the site of exposure to the site where the virus causes fatal encephalitis—that is to say, the "pathogenesis" of airborne rabies infection would have to be studied. In the late 1960s and early 1970s, experiments designed to reveal the pathogenesis of airborne rabies were conducted.[128]

Two important findings resulted from this research. First, it was demonstrated that an animal's susceptibility to developing sickness as a result of an airborne exposure to the rabies virus was closely related to the amount of viral particles in the aerosols to which that animal was exposed.[129] Rabies has long been considered a "dose-related" disease; in the case of airborne exposures, it was concluded that the quantity of viral particles to which an animal was exposed was the most determinative factor governing the chances the virus would infect that animal and cause

128. Tr. at 1246 (Clark).

129. V. Hronovsky & R. Benda, "Experimental Inhalation Infection of Laboratory Rodents with Rabies Virus," 13 Acta Virol. 193 (1969) (admitted into evidence as one of a packet of documents constituting Exh. G–137 and hereinafter referred to as "Laboratory Rodents").

disease.[130] Second, important evidence was collected concerning the path the virus takes in invading an animal's central nervous system after infection through the airborne route.

It was known that some viruses transmitted through aerosol suspensions can cause encephalitis in the animals they infect after they are inhaled into the nasal region by spreading along the olfactory nerves and enter the olfactory bulb, the region of the brain that controls an animal's sense of smell. Other viruses replicate in the lower respiratory tract and lungs after being inhaled through the mouth and nose before attacking the central nervous system.[131] The results of experiments conducted in the late 1960s and early 1970s involving laboratory animals such as mice, guinea pigs, and rabbits indi-

130. Tr. at 1248–49 (Clark) (discussing "Laboratory Rodents," *supra* note 129); Tr. at 1397 (Shope).

131. Apparently, the viruses of vesicular stomatitis, equine encephalomyelitis, and St. Louis encephalitis invade the olfactory nerves in the nasal mucosa, while the viruses that cause pseudorabies (a neurologic disease of farm and domestic animals) and herpes invade a host through other pathways. A. Sabin, J. Casals–Ariet, & L. Webster, "Localization of Virus and Lesions after Nasal Instillation of Rabies in Mice," 39 J. Bacteriology 67, 67 (admitted into evidence as one of a packet of documents constituting Exh. G–137).

cated that when those animals were infected after an exposure to an airborne suspension of rabies virus, the olfactory region was of primary importance in the spread of the virus to the central nervous system.[132] It was found that the virus attached itself to the olfactory nerves of the exposed animals, reproduced there, and quickly spread to the brains of those animals.[133] It was also concluded that while "[t]he participation of lung tissue as the primary site of virus multiplication in inhalation rabies cannot be excluded[,] ... [the test findings]

132. *See* V. Hronovsky & R. Benda, "Development of Inhalation Rabies Infection in Suckling Guinea Pigs," 13 Acta Virol. 198 (1969) (admitted into evidence as one of a packet of documents constituting Exh. G–137 and hereinafter referred to as "Suckling Guinea Pigs"); V. Hronovsky, "Immunofluorescence Study on the Pathogenesis of Fixed Rabies Virus Respiratory Infection in Suckling Mice," 15 Acta Virol. 58 (1971) (admitted into evidence as one of a packet of documents constituting Exh. G–137 and hereinafter referred to as "Suckling Mice"); *see also* H. Fischman & M. Schaeffer, "Pathogenesis of Experimental Rabies as Revealed by Immunofluorescence," 177 Ann.N.Y.Acad.Sci. 78 (1971) (admitted into evidence as one of a packet of documents constituting Exh. G–137 and hereinafter referred to as "Pathogenesis of Experimental Rabies"); "Airborne Rabies," *supra* note 97, at 119–20; "Pathogenesis," *supra* note 16, at 192–93.

133. "Suckling Guinea Pigs," *supra* note 132, at 201; "Suckling Mice," *supra* note 132, at 62; *see also* "Pathogenesis of Experimental Rabies," *supra* note 132, at 94.

suggest that the pulmonary route of virus spread into the [central nervous system] plays no important role in the pathogenesis of rabies in experimental animals infected by inhalation." [134] Results pointing to the same conclusion were obtained regardless of whether street or fixed viruses were used.[135]

These findings indicated that the pathogenesis of rabies virus transmitted to man through an aerosol exposure might be significantly different than the pathogenesis of the virus when transmitted through animal bites or scratches. As noted above, the rabies virus proliferates primarily in nervous tissue. When a human being is exposed to the virus through the bite of an infected animal, the muscles, connective tissue and nervous tissue at the site of the bite wound will be bathed in blood. If the victim of the bite has been immunized against rabies and as a result has developed rabies antibodies in his blood stream, those antibodies would have the opportunity to neutralize the virus before it invaded the victim's central nervous system.[136]

134. "Suckling Guinea Pigs," *supra* note 132, at 201. This conclusion was based on the observation that rabies viral particles often were not found in the lower respiratory tract and lung tissue until after the central nervous system was involved in viral replication, and that findings of virus in the pulmonary region were erratic and quantitatively low.

135. *See* "Suckling Mice," *supra* note 132, at 62.

136. Tr. at 1396 (Shope).

This is not necessarily the case when an individual is infected through the airborne route. From studies relating to biological warfare conducted by Dr. Arnold Wedum at Fort Detrick, it was known that pre-existing antibodies circulating in the blood stream might not provide protection against aerosol exposure to some viruses.[137] A human's olfactory nerves at one end rise out of the mucosal epithelium cells that line the olfactory region inside the nose and at the other extend directly into the region of the brain known as the olfactory bulb.[138] The olfactory nerves are the only nerves in a vertebrate animal such as man that emanate directly from the brain and are exposed to the air.[139] Because the olfactory nerves are superficial structures, the only protection they have against neurotropic viruses in the environment to which they are exposed is provided by the nasal mucosa.[140] The olfactory nerves do not ordinarily come into significant contact with the blood stream, and thus a virus infecting them would not come into contact

137. Exh. G-125H at 141 (Deposition of Dr. Robert H. Huffaker, February 2, 1983).

138. Tr. at 1395 (Shope); Tr. at 1838-39 (Winkler).

139. Tr. at 1395 (Shope).

140. *See* Tr. at 1100-01 (Hattwick).

with pre-existing antibodies circulating in the blood stream.[141] Consequently, absent the presence of a significant level of antibodies in the mucosa, a virus that infects the olfactory nerves can replicate and travel the short distance to the afflicted individual's brain unimpeded by neutralizing antibodies.[142]

Some but not all vaccines produce antibodies in surface membranes such as the nasal mucosa as well as in the blood stream.[143] In 1976 and 1977, it was not known whether the Lilly vaccine produced surface antibodies in the nasal mucosa sufficient to provide protection in the event of an aerosol exposure to the rabies virus.[144] Defendant Lilly did not undertake investigations concerning the effectiveness of DEV against infection by the airborne route at any point in the 1960s or 1970s, even after reports surfaced concerning the laboratory-related death of the veterinarian in 1973,[145] nor were such studies conducted

141. Tr. at 1396 (Shope).

142. Tr. at 1422–24 (Shope); *see also* Tr. at 1837–41 (Winkler).

143. Tr. at 822 (Tillotson).

144. Tr. at 1030–31 (Hattwick); Tr. at 824 (Tillotson); Tr. at 1343 (Clark); Exh. G–125E at 66 (Deposition of Dr. Rajender Abraham); "Laboratory Worker," *supra* note 120, at 1221.

145. Exh. G–125F at 21 (Deposition of Dr. John T. Anderson).

by other researchers. Despite the uncertainty concerning DEV's effectiveness against infection through the aerosol route, the package insert distributed with DEV was not amended at any time to alert users that the vaccine might not protect against disease in the event of such exposures.[146]

In light of the existing knowledge concerning the peculiar vulnerability of the olfactory nerves to infection and the lack of knowledge concerning the effectiveness of the Lilly vaccine in protecting against aerosol exposures to rabies virus, safe laboratory practice in 1976 and 1977 mandated the avoidance of such exposures. If possible, the creation of aerosols containing rabies virus in the laboratory was to be avoided. If the creation of such aerosols was necessary to the accomplishment of the purposes of a particular study, safe laboratory practice required that the aerosols be physically contained.[147] The physical "containment" of an aerosolized etiologic agent is achieved through the use of one of several methods of separating the environment in which the aerosols are created from the environment to which laboratory workers are exposed. Many of these methods were technically feasible in 1976 and

146. Exh. G-125F at 61 (Anderson Deposition); *see* Exh. 87A (Lilly package insert).

147. Exh. G-125H at 33 (Huffaker Deposition, February 1, 1983).

1977, and in fact were commonly employed. The use of an apparatus known as a "laminar flow containment hood" was probably the most common method of "containing" an etiologic agent. This apparatus, as well as other containment methods, will be discussed in greater detail below.

III. THE EXPERIMENTS WITH THE UNI-GLATT MACHINE

This section of this memorandum-decision and order focuses on those aspects of the research conducted by NYSDOH and CDC scientists regarding wildlife immunization relevant to this lawsuit. The research concerning the aerosol transmission of the rabies virus was performed contemporaneously with studies directed at developing a rabies immunization program of an unprecedented scale. With the substantial reduction of the incidence of rabies in domestic animals and man in the United States in the 1950s and 1960s, the attention of a number of rabies experts turned to the control of the disease in wild animals and commercial livestock. Among the animal species particularly susceptible to acquiring and passing on the disease of rabies are foxes, coyotes, skunks, raccoons, and

bats.[148] Whether eradication of the disease of rabies can be accomplished ultimately will turn on science's ability to control the spread of the rabies virus in these and other species found in the wild.

A. *Wildlife Immunization Research*

In 1973, a report prepared by experts under the auspices of the World Health Organization ("WHO") noted that the "massive reduction" of those species responsible for the perpetuation of the rabies disease within a given ecosystem through extermination programs was "the only method available for wildlife rabies control."[149] Such population reduction programs are effective only in "suitable, limited areas,"[150] and even where possible such programs have adverse environmental and economic effects.[151] The WHO report noted that immunization of wildlife was not

148. World Health Organization Technical Report Series No. 523, WHO Expert Committee on Rabies, Sixth Report, at 12 (Geneva 1973) (admitted into evidence as Lilly Exh. B, and hereinafter referred to as "WHO Expert Report"); Tr. at 2060 (Baer).

149. WHO Expert Report, *supra* note 148, at 39; *see also* J. Lewis, "Control of Rabies Among Terrestrial Wildlife by Population Reduction," 2 *Natural History* at 244 (hereinafter "Population Reduction").

150. WHO Expert Report, *supra* note 148, at 41.

151. "Population Reduction," *supra* note 149, at 255–57.

then practicable, and recommended that "[s]tudies in this direction should ... be intensified.... Immunization could reduce the susceptible host population to a point where no further rabies spread is possible." [152]

One of the earliest experiments concerning wildlife immunization had been conducted between 1961 and 1963 by Dr. George M. Baer of the Center for Disease Control. Baer performed these experiments while he was a visiting epidemiologist at the New York State Department of Health's Griffin Laboratory.[153] Baer attempted the oral vaccination of foxes by shooting a fixed rabies virus strain into the mouths of the animals with a mechanical device.[154] Six of the fourteen foxes vaccinated in this manner experienced rises in serum neutralizing antibodies and survived subsequent challenge with the CVS rabies virus strain.[155] The encouraging results obtained in this experiment and in others

152. WHO Expert Report, *supra* note 148, at 41.

153. Tr. at 1110 (Baer Deposition).

154. Tr. at 98 (Debbie); G. Baer, "Wildlife Vaccination," 2 *Natural History* at 261 (hereinafter "Wildlife Vaccination").

155. "Wildlife Vaccination," *supra* note 154, at 263.

conducted in the 1960s [156] fueled discussion of the possibility that non-domestic animals could be immunized by consuming bait distributed in the wild that would contain live rabies virus.[157] Baer himself became a vocal advocate of this approach to rabies control.[158]

Baer reasoned that if bait containing live rabies virus strain were to be distributed in the field safely and also be effective in producing rabies antibodies in the animals that consumed them, the virus strain used would have to be highly attenuated with respect to a wide range of animal species while maintaining its immunizing capability when highly diluted.[159] Baer believed that it was possible to develop such a strain by

156. *See, e.g.*, Tr. 1790–91 (Winkler).

157. "Wildlife Vaccination," *supra* note 154, at 262.

158. Tr. at 1227–28 (Clark).

159. "Wildlife Vaccination," *supra* note 154, at 262; *see also* W. Winkler, J. Shaddock, & L. Williams, "Oral Rabies Vaccine: Evaluation of its Infectivity in Three Species of Rodents," 104 Amer.J.Epidem. 294 (1976) (admitted into evidence as one of a packet of documents constituting Exh. G–137).

further modifying a licensed modified live virus vaccine known as ERA.[160]

The ERA commercial vaccine was derived from the SAD virus strain, which had been developed at the University of Toronto's Connaught Laboratories ("Connaught").[161] The SAD virus strain had been derived from street virus that had been isolated from a rabid dog in Montgomery, Alabama in 1935 by passaging it over fifty times in the brains of mice.[162] In the early 1960s, scientists at Connaught passaged the SAD virus strain an additional four times in the brains of mice to make a virus strain called SAD–4. Dr. Melvin K. Abelseth and two other researchers at Connaught then passaged the SAD–4 virus strain twenty-five times in tissue culture

160. The designation "ERA" is an acronym representing the initials of the surnames of the scientists who developed it. The last names of those scientists were Ellen, Rocketwicki, and Abelseth. *See* Tr. at 1124 (Baer Deposition). Dr. Melvin Abelseth was NYSDOH's Director of Laboratories at the time Jerome Andrulonis contracted the disease of rabies.

161. Tr. at 522 (Abelseth Deposition); Tr. at 1231 (Clark). "SAD" is an acronym for "Street–Alabama–Dufferin," representing respectively the type of virus from which the strain was derived, the state in which the "street" virus had been isolated, and the division of Connaught Laboratories where the SAD strain was developed.

162. Tr. at 522 (Abelseth Deposition).

taken from the kidneys of hamsters, ten times in embryonated chicken eggs, and an additional six times in tissue culture isolated from the kidneys of pigs.[163] The virus strain that resulted from this passage history was designated ERA.[164] A modified live virus strain, ERA was found to be attenuated for dogs, cats, horses, cattle, sheep, and swine, and in the 1960s was licensed for use as a modified live virus vaccine for those species.[165] Thereafter, ERA, which usually attains titers ranging from 3.5 to 4.5, was widely used as a commercial vaccine.

163. Tr. at 522–23 (Abelseth Deposition); "Rabies Virus Strains," *supra* note 52, at 179.

164. Tr. at 523 (Abelseth Deposition).

165. "Rabies Virus Strains," *supra* note 52, at 179; "Canine and Feline Vaccines," *supra* note 43, at 181–82. In some of the scientific literature admitted into evidence in this case, the designations "ERA" and "SAD" are used interchangeably. In Canada, where the vaccine was first licensed, it was designated "ERA." When the vaccine was licensed in the United States, however, it was discovered that "ERA" was a protected trade name, and thus the vaccine was given the name of the strain from which it had been derived, "SAD." *See* Tr. at 1855–56. To avoid confusion, the court will refer to the vaccine developed by Abelseth and others as "ERA," and allusions to "SAD" will refer to the virus strain from which ERA was derived.

The ERA vaccine is the first rabies virus strain discussed in this opinion that was developed in part through passage in animal tissue cell culture rather than wholly through passage in live animals or embryonated eggs. Virus strains adapted to cell systems generally replicate readily, growing large quantities of virus during passaging. After modern concentration and purification techniques are employed, virus strains grown in cell systems yield higher titers of "pure" virus (virus preparations free of non-viral cellular impurities) than can be obtained from the brains of infected animals after passage, concentration, and purification.[166] For all of its advantages, however, virus strains adapted to tissue culture did increase one hazard associated with the laboratory use of rabies virus strains: studies had indicated that such strains were more effective at inducing the disease of rabies after inhalation than strains of identical origin grown in mouse brains.[167]

The ERA vaccine was considered particularly well-adapted for growth in cell culture, rendering the highest yields of "pure" virus of all of the cell culture-adapted

166. "Growth in Cell Culture," *supra* __ at 155–56; (hereinafter "Growth in Cell Culture"); "Concentration and Purification," *supra* note __, at 125.

167. Tr. at 2315–16 (Baer).

strains available in the 1960s and 1970s.[168] Moreover, a virus strain derived from the ERA vaccine tended to have a high "specific infectivity." In any rabies virus strain, most of the viral particles will not penetrate an animal's cells and replicate. As a rule, an unusually high percentage of the viral particles in an ERA-derived strain were capable of infecting an animal's cell system.[169] An ERA-derived strain of virus was considered a strong candidate for use in any wildlife vaccination program in part because of its high infectivity. Baer noted at an early juncture that the immune response in animals injected with the commercial ERA vaccine "apparently result[ed] from multiplication of virus" in the animal to which the vaccine is administered.[170] Because the live virus in the vaccine evidently replicated without spreading to the central nervous system and causing disease in animals for which the vaccine was attenuated, it retained its immunizing ability

168. "Rabies Virus Strains," *supra* note 52, at 179.

169. Tr. at 1232-33 (Clark).

170. G. Baer, M. Abelseth & J. Debbie, "Oral Vaccination of Foxes Against Rabies," 93 Amer. J.Epidem. 487, 487 (1971) (admitted into evidence as Exh. 5 and hereinafter referred to as "Vaccination of Foxes").

when highly diluted.[171] Equally important was the fact that the ERA vaccine was not lethal to foxes, the primary target for the wildlife immunization program that Baer sought to develop because of their role in perpetuating the disease of rabies in the United States.[172] Finally, because the ERA vaccine had proven to be a "safe and effective immunizing agent in six species of domestic animals," [173] it was hoped that a derivative of the commercial vaccine could be developed that would be attenuated for the much wider range of species that could consume baits containing live rabies virus distributed in the wild.

In 1969 or 1970, Baer attempted to produce serum neutralizing antibodies in a grey fox by dropping on the tongue and cheek of the animal one milliliter of ERA strain rabies virus grown on a cell system originating from the kidneys of baby Syrian hamsters and subsequently altered in the laboratory. This cell "line" was known

171. "Vaccination of Foxes," *supra* note 170, at 487.

172. *See* G. Baer, "The Oral Rabies Immunization of Foxes and Dogs with Sausage Baits," 33 Develop.Biol.Standard 417, 418 (1976) (introduced into evidence as one of a packet of documents constituting Exh. G–137 and hereinafter referred to as "Sausage Baits").

173. "Vaccination of Foxes," *supra* note 170, at 487.

as BHK/21.[174] The passaging of the ERA vaccine on BHK/21 cells resulted in a virus strain designated ERA-BHK/21; the ERA-BHK/21 strain administered by Baer in the grey fox experiment had a titer of 6.8.[175] Antibodies were produced. Buoyed by the success of this experiment, Baer ventured north in 1970 to meet with Dr. Abelseth, by this time the Director of Laboratories within NYSDOH's Division of Laboratories and Research, and Dr. John G. Debbie, an NYSDOH scientist who worked directly under Abelseth, in an effort to interest the NYSDOH scientists in his project.[176]

Among the laboratories maintained by the Division of Laboratories and Research was Griffin Laboratory, located in a rural area west of Albany, New York. At Griffin, animal research and diagnostic work related to human disease was performed, and animals were raised for use in experiments conducted in NYSDOH's other laboratories.[177] Dr. Debbie headed the rabies

174. "BHK" is an acronym for "Baby Hamster Kidney;" the number "21" designates a subtype of BHK cells. *See* Tr. at 1124–25 (Baer Deposition).

175. "Vaccination of Foxes," *supra* note 170, at 488; Tr. at 97 (Debbie).

176. Tr. at 97 (Debbie); Tr. at 2288–89 (Baer).

177. Tr. at 91 (Debbie); Tr. at 516 (Abelseth Deposition).

laboratory at Griffin.[178] Debbie was responsible for assuring that all procedures performed in that laboratory were conducted safely, in accordance with good laboratory practice as recognized by the scientific community at the time.[179] Among those working under Debbie was Jerome Andrulonis, at the time a bacteriologist with NYSDOH.[180]

At the 1970 meeting with Abelseth and Debbie, Baer related the promising results obtained in his grey fox experiment, and it was then decided that because NYSDOH had access to foxes for experimental use, further research concerning the oral vaccination of wildlife would be pursued at Griffin.[181] Funding for this study was provided by the State of New York, not by the CDC. The virus strain used in this study

178. Tr. at 91 (Debbie). At Griffin, the rabies laboratory actually encompassed a number of rooms in two buildings. As used in the text, references to Griffin's "rabies laboratory" designates the room in the main building at Griffin where the experiments with the Uni-Glatt machine took place. This room was sizable, measuring approximately thirty feet long and twenty-two feet deep, and was located on the second floor of the main building at Griffin.

179. Tr. at 385 (Debbie); Exh. G-125H at 9 (Huffaker Deposition, February 1, 1983).

180. Tr. at 92-95 (Debbie).

181. Tr. at 99-100 (Debbie).

was supplied from the CDC by Dr. Baer.[182] The CDC commonly prepares and supplies various viral strains, including strains of rabies virus, to approved private and state operated laboratories for use in research projects.[183] The CDC did not in the 1970s and does not now make routine safety inspections of the laboratories to which etiologic agents are supplied.[184]

For the first tests performed at Griffin, Baer supplied Debbie with an ERA virus strain grown on BHK/21 cells. This virus strain had a titer of 7.0. Debbie administered the virus in liquid form to the tongues and cheeks of six red foxes.[185] Andrulonis aided Debbie in this procedure.[186] All six foxes developed serum-neutralizing antibodies, and survived a challenge from a street virus that killed four out of five foxes in a control group.[187] A short time thereafter Debbie repeated the experiment using the ERA vaccine that

182. Tr. at 103–04 (Debbie).

183. Tr. at 2051–53 (Baer).

184. Tr. at 2069–70 (Baer); Tr. at 1814 (Winkler); Tr. at 1953–54 (Testimony of Dr. John H. Richardson).

185. Tr. at 101–04 (Debbie); "Vaccination of Foxes," *supra* note 170, at 488.

186. Tr. at 327–28 (Debbie).

187. "Vaccination of Foxes," *supra* note 170, at 488–89.

had been licensed for commercial use. The vaccine used had a titer of no greater than 3.7. A significant percentage of the foxes which were administered the commercial ERA vaccine developed antibodies and survived challenge.[188] Again, Andrulonis assisted.[189]

Encouraged by the successful results obtained in these experiments, researchers from the CDC and NYSDOH sought to determine what effect an ERA-derived virus strain would have on animal species other than foxes. The researchers wanted to determine whether immune responses could be triggered by an ERA-derived strain in other wildlife species. More importantly, it was necessary to determine whether other animals which might ingest a bait containing an ERA virus in the wild would develop the disease of rabies as a result, since a rabies virus that is attenuated for one species might be pathogenic to another. The researchers were also interested in determining whether a liquid containing live rabies virus could be passed on to foxes and other species in a bait of some sort.[190] The survival of the virus under field conditions was problematic, and thus

188. Tr. at 106–08 (Debbie); J. Debbie, M. Abelseth & G. Baer, "The Use of Commercially Available Vaccines for the Oral Vaccination of Foxes Against Rabies," 96 Amer.J.Epidem. 231 (1972) (admitted into evidence as Exh. G–89).

189. Tr. at 327–28 (Debbie).

190. Tr. at 108–09 (Debbie).

the development of a bait suitably protective of the virus was another focus of research.[191] Research conducted at Griffin investigating these areas of concern was supported in part by a grant from the CDC,[192] although the CDC did not direct the day-to-day operations of the project, devise protocol for the work, or otherwise control the manner in which the work conducted at Griffin was to be performed.[193]

Subsequent attempts to orally vaccinate skunks and raccoons at Griffin with an ERA-derived virus strain, as well as efforts to produce antibodies in mongooses administered an ERA-derived virus strain at the CDC laboratories in Lawrenceville, were not as successful as the experiments involving the oral vaccination of foxes.[194] Foxes apparently can be immunized by an attenuated virus strain that invades through the lingual and buccal mucous

191. "Wildlife Vaccination," *supra* note 154, at 265.

192. Tr. at 115 (Debbie); J. Debbie, "Use of Inoculated Eggs as a Vehicle for the Oral Rabies Vaccination of Red Foxes," 9 Infection & Immunity at 683 (1974) (admitted into evidence as Exh. 6 and hereinafter referred to as "Use of Inoculated Eggs").

193. Tr. at 334 (Debbie).

194. Tr. at 143 (Debbie).

membranes of the tongue and cheek.[195] These other animals tested, however, evidently were not susceptible to infection by the same route.[196] Moreover, it was believed that the modified live rabies virus that was being administered to the animals was being killed by the acids of the animals' stomachs.[197] Consequently, the attention of the researchers at Griffin turned to the development of a method of vaccination through the so-called "enteric route," whereby the virus would be absorbed through the intestines of the animals that ingested it rather than through the tongue and cheek.[198] This would be accomplished by applying an "enteric coating"[199] to a live virus which would protect the virus from the stomach acids of the animal consuming it but would disintegrate once it reached the animal's intestine, allowing the

195. *See* "Sausage Baits," *supra* note 172, at 417–18.

196. *See* Tr. at 2055–56 (Baer).

197. Tr. at 143 (Debbie); Tr. at 2335 (Baer).

198. Tr. at 532–33 (Abelseth Deposition); Tr. at 2055 (Baer); Tr. at 143 (Debbie).

199. This term usually refers to a coating applied to tablets or capsules which prevents release and absorption of the material contained in the tablet or capsule until it reaches the intestine of the human or animal ingesting it.

virus to multiply there and induce an antibody response.[200]

Initially, Debbie applied enteric coating to gelatin capsules containing rabies virus that had been stabilized through a process known as "lyophilization"[201] and administered the capsules to foxes. The capsules proved too large, however, and failed to pass out of the stomachs of the foxes and into their intestines.[202] In or around September 1975, Debbie attended a WHO-sponsored collaborative meeting of scientists, including Baer, concerned with the oral vaccination of wildlife. At this meeting, Debbie discussed this enteric coating experiment in informal presentations. It also appears that Debbie spoke with Baer of the problems he was having with the size of the capsules he had prepared, since Baer subsequently sent Debbie a copy of a patent that had been obtained for an enteric coated capsule containing a modified live virus intended to create antibodies for a contagious viral disease that attacked

200. Tr. at 533 (Abelseth Deposition); Tr. 2055 (Baer).

201. Lyophilization of a biological substance involves the rapid freezing of the substance and the dehydration of the resulting frozen product under high vacuum. This process renders a stable preparation of the biological substance so processed.

202. Tr. at 145 (Debbie).

swine.[203] The patent described a process through which a virus was coated onto processed sugar nonpareils with diameters measuring less than 2.5 millimeters. The virus-coated nonpareils were then enterically coated with a compound of alcohol and an acetic acid known as cellulose acetate phthalate. Debbie sought more information concerning the enteric coating process alluded to in the patent, and was advised by someone at the Albany School of Pharmacy to communicate with the coating department at the WARF Institute, Inc., located in Middleton, Wisconsin.[204]

Debbie contacted a representative of WARF and sought information about the available coating techniques that might be used to create a smaller capsule containing a live rabies virus.[205] In response, Debbie received a letter from Thomas M. Hinkes, the director of WARF's coating department, and an enclosure describing the "Wurster air suspension coating process," which will be discussed in detail below. In his letter, Hinkes indicated to Debbie that "[a]s we agreed, I will check with people at WARF Institute regarding the safety of

203. Tr. at 143–44, 146 (Debbie); *see* Exh. 13 (U.S. Patent No. 3,823,228 (Patented July 9, 1974)).

204. Tr. at 341 (Debbie).

205. Tr. at 146–47 (Debbie).

handling the vaccine, and you in turn will contact the parties in Atlanta, Georgia," [206] those parties being Drs. Baer and Winkler of the CDC.[207]

At some point, Debbie contacted Baer and Winkler, seeking assurances about the safety of the ERA rabies virus and its derivatives. Baer responded with a letter addressed to Debbie dated November 7, 1975, which included the following passage:

> ERA rabies vaccine appears to be one of the most attenuated animal rabies vaccines available. We have used it in this laboratory for years, not only as a commercial product but also as a concentrated BHK tissue culture supernate, with a titer 100 to 1000 times the original vaccine. In working with the material we take the usual laboratory precautions, primarily having those persons in direct contact with the vaccine vaccinated against rabies.[208]

A copy of Baer's letter was forwarded to Hinkes. Shortly after forwarding Baer's letter to WARF, Debbie received a letter from Dr. Winkler. Unlike Baer, Winkler confined his discussion to the commercial ERA vaccine, and did not discuss deriva-

206. Exh. 14.

207. Tr. at 148 (Debbie).

208. Exh. 20.

tives of the vaccine. In his letter, dated December 3, 1975, Winkler related that there was limited data regarding the dangers posed by the ERA vaccine to man, but that the accidental exposures to the vaccine that had been recorded did not result in "problems of any kind." Further, animal studies indicated that the ERA vaccine was "at least as safe" as another animal rabies vaccine that had proven to be "innocuous in man." [209] This opinion was also related to

209. The important passage in Winkler's letter is as follows:

> In response to your query regarding CDC's stance on the safety of [modified live virus] rabies vaccines particularly the ERA strain in laboratory use where human exposure might occur. We do not consider ERA, LEP, or HEP strains of virus hazardous to man. As you know, there is a wealth of empirical data indicating that the LEP strain is innocuous for man; the HEP strain has been used in experimental inoculation studies in man, again without difficulty; there is only limited information on the accidental inoculation of ERA strain into man. We are currently accumulating data on ERA exposures in man and of over 30 persons exposed in the last 18 months there have been no problems of any kind associated with the exposures. There are numerous studies defining the safety of ERA in animals and when these data are compared with similar results for LEP or HEP, the ERA vaccine appears to be at least as safe as LEP.

Exh. 21. The "LEP" virus strain referred to in the letter is a licensed vaccine for use in dogs. "LEP" is an acronym for "Low Egg Passage."

WARF officials.[210] Whatever apprehensions WARF officials had concerning the project were apparently mollified: arrangements were made for a demonstration of the Wurster air suspension process to be conducted by a WARF representative at Griffin.[211]

The HEP ("High Egg Passage") strain was derived from LEP, and is a licensed vaccine for dogs, cats, and cows. The LEP strain, to which the ERA vaccine was directly compared in the above-quoted letter, was believed to be non-pathogenic to man in part because of experimentation conducted in the late 1940s and early 1950s involving terminally ill cancer patients, who apparently did not die from rabies after being injected with solutions containing that virus strain. *See* Tr. at 2140–41 (Baer); Tr. at 1785 (Winkler).

210. Tr. at 156 (Debbie).

211. *See* Tr. at 157–60 (Debbie); Exh. 27 (Memorandum from Dr. John G. Debbie to Dr. David Axelrod dated February 11, 1976); Exh. 28A (Memorandum from Dr. Debbie to Dr. Axelrod dated March 18, 1976).

B. *The Initial Uni-Glatt Experiments*

On March 30, 1976, WARF representative Harlen Hall arrived at Griffin to demonstrate how a machine known as the Uni-Glatt could be used to coat sugar nonpareils first with a modified live rabies virus strain and then with an enteric coating.[212] The Uni-Glatt machine, which effectuated the air suspension coating process developed by Dale Wurster, was manufactured by defendant Glatt GmbH and distributed by defendant Glatt Air Techniques, Inc. Debbie was sufficiently impressed with the machine to use it on three occasions subsequent to the Hall demonstration in attempts to develop an enterically coated pill containing live rabies virus. The last Uni-Glatt experiments performed at Griffin were conducted on March 29 and 30, 1977. Funding for the Uni-Glatt experiments conducted after the Hall demonstration was provided by the CDC, in exchange for which Debbie was to provide the CDC with some of the coated nonpareils prepared in the course of the Uni-Glatt experiments.[213] Other than providing the funds for the enteric coating experiments, however, officials from the CDC were not directly in-

212. Tr. at 160 (Debbie); Exh. 27.

213. This funding arrangement is described below, *infra* at note 427.

volved in the manner in which the Uni-Glatt experiments were performed, nor did they seek to control the means through which those experiments were carried out.[214]

The Uni-Glatt machine[215] was designed to suspend solid pellets or pills in a column of upflowing air so that coating materials could be sprayed onto them.[216] It was commonly used for the sugarcoating of candy or pharmaceutical products.[217] The Uni-Glatt consisted of a console which was approximately three feet in length, two and

214. Tr. at 2061-62, 2065-67 (Baer); *see also* Exh. G-133 (Answers to Interrogatories of the Defendant United States of America to the Third-Party Defendant New York State Department of Health, dated April 19, 1985).

215. The Uni-Glatt machine is pictured in a photograph admitted into evidence as Exh. 51. An enlarged copy of this photograph was admitted into evidence as Exh. 52, and a series of large-scale close-up photographs of the Uni-Glatt were admitted into evidence as Exhs. 53A-O. The latter series of photographs represent the Uni-Glatt machine as it appeared on March 29, 1977, the date that the rabies virus invaded the body of Jerome Andrulonis, *see* text, *infra* at 1471, and as it appeared in June 1977 when smoke tests were performed by George Mallison of the CDC that demonstrated that the machine was not airtight. Tr. at 211-12 (Debbie).

216. Tr. at 162 (Debbie).

217. *See* Exh. G-125D at 156-57 (Deposition of Dr. C. David Fox).

one-half to three feet in depth, and about two and one-half feet in height, a large pressurized air tank, a long transparent column that sat atop the console, a peristaltic pump that was attachable to a valve located at the bottom of the column, and a long flexible exhaust duct that was attachable to the top of the machine.[218]

The machine's console had gauges and control dials on its front, and inside it was a labyrinth of tubes and switches which enabled the machine to suspend solid materials within the transparent column.[219] Some of those tubes led to the large air tank which supplied pressurized air to the bottom of the console when the machine was in operation.[220] The column, which was comprised of two sections of rigid, transparent plastic tubing, fit into an opening on the top of the console.[221] Approximately one-third of the length of the col-

218. Tr. at 162–63 (Debbie); Exh. 52. The machine that was used in Harlen Hall's demonstration had an exhaust duct that connected to the back of the console rather than the top of the cylindrical column. On the machines that were used at Griffin subsequent to this initial demonstration, the exhaust duct connected to the top of the column. Tr. at 182 (Debbie).

219. Exhs. 53A, 53B, 53C; Tr. at 287–89 (Debbie).

220. Tr. at 172 (Debbie).

221. Exh. 53E; Tr. at 291 (Debbie); *see* Memorandum of George F. Mallison, Assistant Director of Bacterial Diseases Division at CDC, dated September 27, 1977, at 2 (admitted into evidence as Exh. 66, and hereinafter referred to as the "Mallison Report").

umn would fit below the surface of the console, while the remainder protruded upward.[222] The lower part of the column was conical in shape, increasing in diameter from the surface of the console upward. The upper section of the column was cylindrical, and was attached to the conical section by a metal collar.[223] A cloth filter was placed over the top of the column and was held fast by a metal ring, allowing air as well as other gasses to be passed through and vented out of the machine but preventing the solid materials suspended within the column from escaping.[224] An opaque, sleevelike cap was placed over the filter and secured to the top of the column, and the exhaust duct ran out of the top of this cap.[225]

The exhaust duct was expected to direct the air expended from the Uni-Glatt out of the laboratory in which the machine was operated.[226] However, the machine operated under positive pressure, with the air pressure within the machine's column becoming greater than that outside the machine once the machine was activated.

222. Compare Exh. 52 with Exh. 53H.

223. Exh. G-125I at 107-08 (Deposition of George F. Mallison); *see* Exh. 53H.

224. Tr. at 292-93 (Debbie); Exh. 53G.

225. *See* Exhs. 53N, 53O; Tr. at 300-01 (Debbie).

226. *See* Tr. at 170 (Debbie).

Consequently, any opening to the external environment would be the path of escape for gases and other airborne suspensions within the machine.[227] Since the Uni-Glatt machine was not airtight, leaks from the machine were forced out into the atmosphere surrounding it. A substantial leak of air occurred where the metal collar joined the conical and cylindrical sections of the transparent column and where the opaque cap was fitted onto the top of the column.[228] Despite this permeability, the Uni-Glatt was never operated at Griffin within any of the various physical containment systems that were available in 1976 and 1977,[229] and thus anyone working in close proximity to the machine while it was in operation was exposed to any aerosols created within the Uni-Glatt's column.

On March 30, 1976, Harlen Hall met Debbie and his assistants, including Andrulonis, at Griffin's rabies laboratory. An unassembled Uni-Glatt machine had been transported to the laboratory by two of Debbie's assistants. Hall first showed the NYSDOH employees how to assemble the ma-

227. Exh. G-125D at 61–62 (Fox Deposition).

228. Mallison Report, *supra* note 221, at 2; Exh. G-125I at 202–03 (Mallison Deposition); Tr. at 187, 304–05 (Debbie).

229. *See* text, *infra* at 1478–1479 and note 411.

chine.[230] The Uni-Glatt was placed on a table near a window of the laboratory, and the exhaust duct ran from the machine out the window.[231] Hall then conducted a test "run" of the machine. Spherical sugar nonpareils with a diameter of approximately two millimeters were blown upward and suspended in the Uni-Glatt's transparent column. On the initial demonstration run, a rabies virus strain developed by passaging ERA vaccine twice in the brains of suckling mice ("ERA-SMB") was siphoned from a one-liter flask by the peristaltic pump into the valve at the bottom of the transparent column and sprayed through a nozzle upward into the column and onto the air-suspended nonpareils.[232] The process created an aerosol containing live viral particles of the virus strain used. The nonpareils would darken in color as the virus solution was sprayed onto them.[233] The aerosolized virus solution itself, though colorless like water,[234] could be visualized

230. Tr. at 171-72 (Debbie).

231. Tr. at 170, 364 (Debbie); Exh. G-125B at 380-81 (Deposition of Alan Grzelecki).

232. Tr. at 171-72, 177-78, 349 (Debbie); Exh. G-125C at 77-78 (Trimarchi Deposition); *see* Mallison Report, *supra* note 221, at 2-3.

233. Tr. at 2127 (Baer); Tr. at 1220-21 (Baer Deposition).

234. Tr. at 351 (Debbie).

within the column as it was being sprayed onto the nonpareils.[235] Leakage of the virus aerosol from the Uni-Glatt into the atmosphere of the laboratory, however, apparently was not visible to the unaided eye and left no trace outside the machine.[236]

Nonetheless, on occasion nonpareils would leak out of the machine and into the laboratory environment.[237]

Once the virus solution had been sprayed upon the nonpareils, the Uni-Glatt machine was disassembled and the nonpareils were removed and sifted to break up clumps.[238] The machine was then reassembled and a flask containing water soluble 5% methylcellulose was attached to the machine's peristaltic pump. The nonpareils were again suspended by upward air flow in the column, and the methylcellulose solution was sprayed onto the virus-coated nonpareils.[239]

235. Tr. at 481 (Debbie).

236. Tr. at 351 (Debbie); Tr. at 540 (Abelseth Deposition); Tr. at 2128, 2263 (Baer).

237. Tr. at 181 (Debbie); Tr. at 540–41 (Abelseth Deposition).

238. Tr. at 185 (Debbie); Mallison Report, *supra* note 221, at 2.

239. Tr. at 185, 350 (Debbie); Mallison Report, *supra* note 221, at 2.

This coating was intended to protect the virus from a third coating that was applied to the nonpareils consisting of hydroxy methylcellulose phthalate, a cellulose dissolved in a volatile ethyl alcohol solvent.[240] After the second coating had been sprayed onto the nonpareils, the machine was again disassembled and the nonpareils were sifted.[241] The third coating, which was intended to protect the virus from the stomach acids of animals that would subsequently consume baits containing the nonpareils, was then applied. The application of this cellulose solution to the nonpareils created a reddish dust that leaked out of the Uni-Glatt machine during this part of the coating process. The leak was readily perceptible to the human eye.[242]

These three coating procedures together constituted a "run," and it took between four and six hours to complete a run.[243] The sugar nonpareils tended to stick together as they were being suspended within the Uni-Glatt's transparent column, frustrating effective application of the material being sprayed onto them. On occasion, the machine would be stopped in the middle of one of the three phases of a

240. Tr. at 350–51 (Debbie).

241. Tr. at 185–86 (Debbie).

242. Tr. at 185–86, 350 (Debbie).

243. Tr. at 187, 357–58 (Debbie); Mallison Report, *supra* note 221, at 3.

coating run and disassembled so that clumps of nonpareils could be broken up manually.[244] Moreover, while the machine was in operation, it was necessary to watch the nonpareils constantly. A laboratory worker, usually Debbie or Andrulonis, would sit close to the machine while it was in operation and tap the transparent column with the handle of a screwdriver to prevent the nonpareils from sticking together.[245] Andrulonis was masked and gloved when he performed this function.[246] Commonly, Andrulonis would place his face within six to twelve inches of the column while tapping it, and would remain that close to the Uni-Glatt machine for minutes at a time. This task was performed repeatedly over the course of a run.[247]

Because the Uni-Glatt was not airtight and was not operated within a physical containment system, Andrulonis experienced a substantial aerosol exposure to the virus strains that were propelled through the machine when he was monitoring the nonpareils in this manner. After the initial demonstration run, moreover, the researchers at NYSDOH knew that the machine was not airtight. This was evident from

244. Mallison Report, *supra* note 221, at 3.

245. Tr. at 185, 411-12 (Debbie).

246. Tr. at 553 (Abelseth Deposition).

247. Tr. at 185, 412 (Debbie); *see also* Tr. at 2119 (Baer); Exh. G-1251 at 113 (Mallison Deposition).

the reddish dust deposited around the machine during the third phase of a coating run, as well as from the fact that nonpareils occasionally escaped from the machine's cylinder.[248] Even so, the only precautions taken by the NYSDOH workers during the subsequent Uni-Glatt experiments were the use of masks and gloves when working around the machine and the maintenance of a high level of antibodies in their blood system through periodic inoculations with the Lilly vaccine.[249] Dr. Debbie's testimony indicated that he believed that an individual with a sufficiently high antibody titer would be protected against

248. Tr. at 187, 413–14 (Debbie).

249. *See* Tr. at 452–57 (Debbie). After the case of the veterinarian who contracted rabies in a laboratory in Texas was reported in 1973, the researchers working with the rabies virus at Griffin adopted an unofficial policy of closely monitoring the antibody titers of individuals who were exposed to the virus in their work. Tr. at 203–04 (Debbie). Serum-neutralizing tests were regularly performed every six to nine months, and booster shots were given whenever an individual's antibody titer was measured at less than 1:8. Tr. at 204 (Debbie). The scientists at Griffin always used the rabies vaccine manufactured by defendant Lilly and distributed by defendant Thompson & Sons. Tr. at 208 (Debbie).

any laboratory exposure to the rabies virus.[250]

Debbie remained in fairly constant contact with Baer after the first use of the Uni-Glatt in March 1976, conferring with Baer once or twice a month.[251] Debbie testified that at some point after the March 1976 demonstration run and before the final Uni-Glatt experiments conducted in March 1977 he told Baer of the reddish dust that was deposited about the laboratory during the third phase of the coating run.[252] Baer denied this, testifying that Debbie did not tell him of the dust before the March 29, 1977 Uni-Glatt experiment. Baer claimed that he did not learn that the Uni-Glatt was not airtight until he became aware of the results of tests that were conducted on the machine approximately three months after the March 29 experiment.[253] For reasons set out in detail below,[254] the court accepts Debbie's testimony and rejects that of Dr. Baer.

250. Tr. at 457 (Debbie); *see also* Tr. at 384 (Debbie).

251. Tr. at 2054–55 (Baer).

252. Tr. at 504 (Debbie); *see also* Tr. at 481–83 (Debbie).

253. Tr. at 2132–33 (Baer); *see also* Tr. at 2283–86, 2352–53 (Baer). The tests in question were performed by Dr. George F. Mallison of the CDC, and the results of those tests were reported in the Mallison Report, *supra* note 221.

254. *See* text, *infra* at 1491.

During the initial demonstration run conducted with Hall present in March 1976, the NYSDOH investigators used an ERA-SMB virus strain prepared by Andrulonis at Griffin. Prior to coating, the virus strain had a titer of approximately 6.[255] As the virus was propelled through the machine and sprayed onto the nonpareils, the heat generated by the machine killed most of the viral particles.[256] After the virus strain had gone through the three-stage coating process, its titer had been reduced to approximately 1.[257] Debbie fed the coated nonpareils to laboratory animals kept at Griffin, including foxes, skunks, and raccoons, and was able to develop only an

255. Tr. at 172, 368 (Debbie).

256. Tr. at 179–80 (Debbie). At one point during the demonstration run, the researchers attempted to reduce the heat of the machine by removing a side panel of the console and placing an ordinary household fan on a chair to blow air into the console. Tr. at 179, 288–89 (Debbie); *see* Exh. 53B. This practice was repeated during other experiments involving the Uni-Glatt. On at least one occasion, ice packs were placed at the base of the machine's column in an effort to lower the temperature of the Uni-Glatt when it was in operation. Exh. G-125A at 313–14 (Deposition of Robert J. Rudd); Exh. G-125C at 78 (Trimarchi Deposition). It does not appear that either method noticeably diminished the heat generated by the machine during the coating run.

257. Tr. at 368–69 (Debbie).

irregular and uneven level of serum antibodies to rabies in those animals.[258] Similarly sporadic results were obtained in dogs and mongooses which were fed the coated nonpareils at the CDC's laboratories in Georgia.[259]

Another Uni-Glatt experiment was conducted at Griffin in July 1976. Once again, an ERA-SMB virus strain with a titer of 6 was used. As in the initial experiment involving the Uni-Glatt, the end-product had a greatly reduced titer, thereby limiting its effectiveness at producing rabies antibodies in animals that consumed it.[260] The coating experiment was repeated in September 1976, and this time two coating runs were completed, one using an ERA-BHK/21 virus strain obtained from the CDC which had a titer of approximately 5.5, and one using an ERA-SMB with a titer of approximately 6. A substantial loss of titer occurred during the course of the coating process in both runs.[261]

In evaluating the results obtained from these experiments, Debbie reasoned that if a virus solution with a higher titer was used, an end-product with a sufficiently

258. Tr. at 366 (Debbie); Tr. at 2064 (Baer).

259. Tr. at 2064 (Baer).

260. Tr. at 368–69 (Debbie).

261. Tr. at 371 (Debbie).

high titer to effectively produce antibodies in animals could be obtained, even if the heat generated by the Uni-Glatt machine during the coating process killed most of the virus.[262] The researchers at Griffin, however, were unable to obtain a titer much higher than 6, the titer that had been achieved in the ERA-SMB strains which they had prepared for the earlier Uni-Glatt experiments. This titer level had proved inadequate in three prior coating runs. Debbie again turned to Dr. Baer in Georgia, who agreed to prepare a rabies virus strain with a higher titer than could be obtained by the workers at NYSDOH.[263]

C. *The Virus Strain Prepared by Dr. Baer and Used in the March 29, 1977 Uni-Glatt Experiment*

To prepare a high titer virus strain, Baer chose a virus strain that had been derived from the ERA vaccine and had been passaged in BHK/21 tissue culture numerous times. Baer passaged the virus an additional time in the BHK/21 cell system.[264] The result was a virus strain several passages removed from the ERA commercial vaccine from which it was derived.

262. Tr. at 2372-73 (Baer).

263. Tr. at 191 (Debbie); Tr. at 2372-73 (Baer).

264. Tr. at 2343-46 (Baer).

It was not surprising that Baer chose an ERA-BHK/21 virus strain for use in the Uni-Glatt experiments. By the 1970s, the BHK/21 cell line had become a favorite medium for the preparation of rabies virus strains because of its "extreme susceptibility to the virus."[265] The BHK/21 cell system tended to yield high quantities of viral particles when a virus strain was grown on it, particularly when the virus strain had a prior history of passage on animal tissue cell culture, as did the ERA vaccine.[266] Moreover, there was evidence indicating that an ERA-BHK/21 virus strain would have a greater ability to withstand the heat generated by the Uni-Glatt machine. In the early 1970s, an ERA virus strain passaged four times on BHK/21 cells was found to be very efficient at replicating at comparatively high temperatures, while remaining less pathogenic, though still lethal, when inoculated intracerebrally into suckling mice than other strains which were not

265. "Growth in Cell Culture," *supra* note 78, at 158.

266. "Growth in Cell Culture," *supra* note 78, at 171-72.

capable of reproducing at higher temperatures.[267]

Notwithstanding the perceived advantages associated with the ERA-BHK/21 virus strain, relatively little was known about it in March 1977. The ERA vaccine virus strain had been derived through passage in the cell systems of three animal species—hamsters, chickens, and pigs. In creating ERA-BHK/21, additional passages on an altered cell system originating from baby Syrian hamsters were performed. Any time a virus strain is passaged, the potential to change its characteristics exists, and this change is fairly unpredictable: the virus may become more or less pathogenic for a specific species after it has been passaged.[268] The changes resulting from the mutations that occur during passaging are particularly unpredictable when a virus strain adapted to the

267. H Clark & T. Wiktor, "Temperature-Sensitivity Characteristics Distinguishing Substrains of Fixed Rabies Virus: Lack of Correlation with Plaque-Size Markers on Virulence for Mice," 125 J.Infec.Dis. 637, 638–41 (1972) (admitted into evidence as one of a packet of documents constituting Exh. G-137).

268. Tr. at 1793-94 (Winkler); *see also* Tr. at 1855 (Winkler) (By passaging a virus strain, "[y]ou've made an unknown animal out of it.").

cell system of one species is passaged in cells isolated from another species.[269]

By March 1977, a handful of studies involving the ERA-BHK/21 virus strain had been conducted. The results of those studies were mixed. In 1975, it was reported that safety testing that had been undertaken indicated that the ERA-BHK/21 strain "would probably be non-pathogenic" for red foxes if ingested orally.[270] The titers of the virus strains tested ranged from 6.5 to 7.0.[271] However, ERA-BHK/21 virus strains with similar titers produced disease in white mice, Norway rats, and cotton rats when fed to animals of those species either in liquid form or

269. Tr. at 1427 (Shope); *see also* G. Bijlenga & L. Joubert, "High Pathogenicity for Mice by Oral Inoculation of a Viral Component in Live Modified Rabies Vaccine ERA/BHK," 47 Bull. Fr.Vet.Acad. 423 (1974) (translation) (admitted into evidence as one of a packet of documents constituting Exh. G-137 and hereinafter referred to as "High Pathogenicity for Mice").

270. W. Winkler, R. McLean, & J. Cowart, "Vaccination of Foxes Against Rabies Using Ingested Baits," 11 J. Wildlife Diseases 382, 387 (1975) (admitted into evidence as Exh. G-90 and hereinafter referred to as "Ingested Baits").

271. "Ingested Baits," *supra* note 270, at 382.

after incorporation into sausage baits.[272] In an experiment conducted by Dr. Baer, solutions containing ERA-BHK strains were injected into the cerebrospinal fluid of the central nervous systems of rhesus monkeys, which like humans are of the order of primates. An ERA-BHK strain is similar to an ERA-BHK/21 strain except that the former is prepared on baby hamster kidney cell lines as they are configured in nature, while the latter is prepared on altered cell lines. The ERA-BHK virus strains used in Baer's experiment had titers ranging from 4.5 to 8.5 and death resulted in virtually all of the monkeys injected.[273] A study conducted in France using the ERA-BHK/21 strain and another strain created by passaging ERA-BHK/21 in another cell line indicated that a highly pathogenic strain of virus could result when the ERA-BHK/21 strain was allowed to mutate in a cell line

272. W. Winkler, J. Shaddock, & L. Williams, "Oral Rabies Vaccine: Evaluation of its Infectivity in Three Species of Rodents," 104 Amer.J.Epidem. 294 (1976) (admitted into evidence as part of a packet of documents constituting Exh. G-137).

273. *See* G. Baer, J. Shaddock, & L. Williams, "Prolonging Morbidity in Rabid Dogs by Intrathecal Injection of Attenuated Rabies Vaccine," 12 Infection & Immunity 98 (1975) (admitted into evidence as Exh. 165); *see also* Tr. at 2326-27 (Baer).

different from that to which it had been adapted.[274] At a symposium held in Atlanta in September 1976, two German investigators reported that the oral administration of ERA-BHK/21 produced disease in Syrian hamsters, brown rats, and field mice.[275] These researchers noted that tests involving other ERA derivative strains revealed a "marked difference in susceptibility of closely related species," making it difficult to assess the range of species for which an ERA-derived virus strain might prove pathogenic.[276]

274. *See generally* "High Pathogenicity for Mice," *supra* note 269; Tr. at 1234–35 (Clark).

275. Tr. at 1236 (Clark); G. Wachendörfer & U. Forster, "Safety Testing of Attenuated Rabies Vaccines in European Wildlife Species" at 2 (1976) (transcript of presentation made at CDC Symposium on Advances in Rabies Research (September 7–9, 1976) (admitted into evidence as part of a packet of documents constituting Exh. G–137 and hereinafter referred to as "Safety Testing of Attenuated Rabies Vaccines"). An abstract of this presentation is reproduced in United States Department of Health, Education, and Welfare, "Symposium on Advances in Rabies Research" at 7 (1976) (admitted into evidence as Exh. G–112).

276. "Safety Testing of Attenuated Rabies Vaccines," *supra* note 275, at 3.

It was not known with certainty whether man was susceptible to developing the disease of rabies as a result of infection by ERA-derived virus strains.[277] The ERA vaccine was widely considered non-pathogenic to man.[278] But with regard to modified live rabies virus strains derived from the ERA commercial vaccine (including ERA-BHK/21), there was "no reliable information on which to judge the risk" that attended human exposure to such strains,

277. Tr. at 1236 (Clark); *see* Tr. at 1829–30 (Winkler).

278. United States Department of Health, Education and Welfare, "Recommendations of the Public Health Service Advisory Committee on Immunization Practices: Rabies," 25 Morbidity and Mortality Weekly Report 403, 406 (1976) (admitted into evidence as Exh. Lilly Y and hereinafter referred to as "MMWR on Rabies Immunization"). The evidence upon which this opinion was based was necessarily limited, however. Because it is ordinarily ethically impermissible to conduct experiments through which human subjects are directly and intentionally exposed to a live rabies virus, *but see supra* note 209, only indirect data gathered from the reported incidents of accidental exposures to the commercial ERA vaccine was available. In 1976, there had been over thirty such reported exposures (eight to ten of which involved individuals who had not been immunized against the rabies virus), and the disease of rabies had not developed in any of the individuals exposed. *See* Exh. 21 (Letter from Dr. William G. Winkler to Dr. John G. Debbie dated December 3, 1975); Tr. at 1786–87, 1848–50 (Winkler); *see also* Tr. at 645–46 (Abelseth Deposition).

and in a 1976 publication the CDC recommended that such virus strains "should be regarded as potentially virulent for purposes of managing the treatment of exposed humans." [279]

While the danger to man posed by any ERA–BHK/21 virus strain was unknown in March 1977, the particular strain prepared by Baer for the Uni–Glatt experiment had an added element of risk: it had been concentrated to an extraordinarily high titer. The virus strain that was ultimately used in the Uni–Glatt experiment conducted March 29, 1977 had been developed by passaging the ERA commercial vaccine in BHK/21 cells between two and twenty-five times, with the virus reaching a titer of 6.7. The virus strain was then concentrated, and the volume of the solution containing the viral particles was reduced from approximately 950 milliliters to approximately seventy-five milliliters. This procedure increased the titer of the virus strain to 8.1.[280] This was the highest titer of any rabies virus strain Baer had transported out of the CDC's laboratories in Georgia.[281] This strain was over one hundred times more pathogenic to mice than any of the

279. "MMWR on Rabies Immunization," *supra* note 278, at 406.

280. Tr. at 2106–07 (Baer).

281. Tr. at 1135 (Baer Deposition).

virus strains used in the earlier Uni-Glatt experiments, and was approximately 10,000 times more pathogenic to mice than the commercial ERA vaccine from which it was derived.[282]

D. *The March 1977 Uni-Glatt Experiments*

Dr. Baer wanted to witness the coating process accomplished through the use of the Uni-Glatt machine first hand, so he decided to personally deliver the high-titered virus strain he had prepared in Georgia to Dr. Debbie in Albany and stay to observe the Uni-Glatt machine in operation.[283] On the evening of March 28, 1977 Baer travelled to Albany by air and was met at the airport by Debbie. Baer had brought the virus strain with him, and that evening he and Debbie took the virus to Griffin and stored it in a laboratory freezer.[284] Baer informed Debbie that the virus

282. As noted above, the titer of a rabies virus strain is expressed in terms of its common logarithm. *See* text, *supra* at 1442. The titer of the ERA-BHK/21 virus strain prepared by Baer (8.1) was two common logarithms greater than that of any of the strains previously used in the Uni-Glatt machine (which never exceeded a titer of 6). The titer of the ERA-BHK/21 virus strain used in the March 29, 1977 experiment was approximately four logarithms greater than the commercial vaccine, whose titer did not usually exceed 4.

283. Tr. at 2077 (Baer).

284. Tr. at 2078 (Baer).

was an ERA-BHK/21 strain and that it had a titer of 8.1.[285] This information was related to the other laboratory workers involved in the Uni-Glatt experiments prior to the experiment conducted the next day.[286] At no time prior to the use of the virus in the Uni-Glatt experiment conducted March 29, 1977 did Baer disclose to Debbie or any other NYSDOH employee how the virus had been prepared, how many times the virus had been passaged in BHK/21 cells, or of any dangers that might inhere in the use of the virus in an experiment in which aerosols containing the virus would be created.[287]

Consistent with ordinary CDC practice, Baer did not conduct a safety inspection of the rabies laboratory at Griffin before the virus he had prepared was supplied to Debbie.[288] As noted above, the CDC did not exercise control over the manner in which the March 29 Uni-Glatt experiment was conceived or carried out. The court finds,

285. Tr. at 2078 (Baer); *see also* Tr. at 372-78 (Debbie). Debbie recalls being told that the virus strain had a titer of 8.5. The court will accept the more conservative recollection of Dr. Baer on this point.

286. Exh. G-125C at 162-63 (Trimarchi Deposition).

287. *See* Tr. at 192 (Debbie); Tr. at 2078 (Baer); *but see* Tr. at 2356-57 (Baer).

288. Tr. at 2069-70 (Baer); *see also* Tr. at 1814 (Winkler); Tr. at 1953-54 (Richardson).

however, that Dr. Baer retained the power to prevent Debbie from using the virus strain he had prepared in the March 29 experiment even after he had surrendered possession of the strain to Debbie on the evening of March 28, and that he could have reclaimed the virus strain at any time prior to its use in the Uni-Glatt experiment conducted that day.[289]

On the morning of March 29, Baer accompanied Debbie to the rabies laboratory at Griffin. The Uni-Glatt machine had already been assembled in the laboratory and had been filled with sugar nonpareils by the time they arrived. Debbie retrieved the ERA-BHK/21 virus solution from the laboratory freezer, and it was set out to thaw.[290] While the virus strain was being prepared for use by Andrulonis, Debbie activated the air currents of the Uni-Glatt and the nonpareils within the machine's transparent column began to circulate.[291]

Baer testified that in this initial period in which the Uni-Glatt was operated—before any rabies virus solution had been introduced into the machine—he observed nonpareils exiting the exhaust duct and entering the laboratory environment, and that Debbie repaired the leak in the duct with

289. Tr. at 2357-58 (Baer).

290. Tr. at 2117-18 (Baer).

291. Tr. at 2118-19 (Baer).

adhesive tape or electrician's tape.[292] This testimony lacks credibility. It was established at trial that a cloth filter placed at the top of the cylindrical portion of the Uni-Glatt's column at the point where the exhaust duct was attached prevented the nonpareils from exiting the column and entering the duct.[293] This filter was not defective, and it did not pose problems during the Uni-Glatt experiment conducted March 29, 1977.[294] Thus it was impossible for Baer to have observed nonpareils escaping from the exhaust duct. Rather, the court concludes that Baer observed the nonpareils escape from the column within which the nonpareils were suspended, and that Baer observed this leakage before any virus solution was aerosolized into the column of the machine.

This initial "warm-up" period in which the Uni-Glatt was operated before any solutions were introduced into it lasted approximately fifteen minutes.[295] During this time, Andrulonis removed a small portion of the virus solution Baer had supplied

292. Tr. at 2128–29, 2275, 2277–83, 2351–53 (Baer).

293. Tr. at 186–87, 292–93 (Debbie); Exh. 53G; *see also* Tr. at 2282–83 (Baer).

294. Tr. at 186–87 (Debbie).

295. Tr. at 2118 (Baer).

so that the titer of the strain used could be verified subsequent to the experiment.[296] The remaining portion of the virus solution was placed in a flask, and the flask was attached to the Uni-Glatt's peristaltic pump.[297] Baer testified that he realized that the virus solution was going to be sprayed onto the nonpareils within the Uni-Glatt's transparent column even before the solution was attached to the pump, and that by forcing the solution through the nozzle at the base of the Uni-Glatt's column an aerosol would be created.[298]

After the flask was attached to the peristaltic pump, the virus solution was slowly introduced into the Uni-Glatt's column, and the upward air current within the column was again activated.[299] The virus was applied to the nonpareils within the column for a little more than an hour.[300] Debbie and Andrulonis worked in close proximity to the machine, adjusting the air flow and the rate at which the aerosolized virus solu-

296. Tr. at 378 (Debbie); Tr. at 2119 (Baer). Special precautions were not taken in handling the virus solution. Tr. at 381-82 (Debbie).

297. Tr. at 2118 (Baer).

298. Tr. at 2350-51 (Baer).

299. Tr. at 2118 (Baer).

300. Tr. at 2129 (Baer).

tion was sprayed onto the nonpareils.[301] From time to time, either Debbie or Andrulonis would tap the column with a screw driver handle to prevent the nonpareils from sticking together and from adhering to the side of the column.[302] Baer witnessed the NYSDOH researchers periodically deactivate the Uni-Glatt and disassemble it in order to sift the nonpareils.[303]

Baer observed most of the coating run in which the ERA-BHK/21 virus strain he provided was incorporated into enterically coated baits.[304] Baer observed the coating procedure closely, and he himself was often within a foot or two of the machine while it was in operation.[305] The following day, after Baer had returned to Georgia, another coating run was completed, this time using an ERA-SMB virus strain with a

301. Tr. at 2118 (Baer).

302. Tr. at 184–85 (Debbie).

303. Tr. at 2279 (Baer). During the March 29 experiment, the machine was also disassembled in order to place an anti-static cloth within the transparent column in an effort to minimize the tendency of the nonpareils to stick together. Tr. at 186, 382 (Debbie). This was the first time anti-static cloths were used in a Uni-Glatt experiment.

304. Tr. at 2126, 2130–31 (Baer).

305. Tr. at 358 (Debbie); Tr. at 2119–20 (Baer).

titer of approximately 5.5 that had been prepared by Andrulonis at Griffin.[306]

As with the previous Uni-Glatt experiments, the Uni-Glatt machine was not operated within a physical containment system of any kind during either of the coating runs conducted in March 1977.[307] The researchers who worked in close proximity to the machine had been immunized against rabies.[308] Specifically, the level of rabies antibodies present in Jerome Andrulonis' blood stream at the time of the March 1977 Uni-Glatt experiments was higher than the minimum that was recommended by the CDC in the Laboratory Safety Manual dis-

306. Exh. G-125C at 162 (Trimarchi Deposition); *see* Tr. at 368-71 (Debbie); Exh. 61 (Handwritten investigation report for New York State Retirement System memorializing information obtained from Dr. John G. Debbie and Charles Trimarchi concerning Andrulonis' exposures to rabies virus in the course of his work) (hereinafter referred to as "NYS Retirement System Report").

307. *See* Tr. at 202-03 (Debbie); *See generally* Mallison Report, *supra* note __.

308. *See* Tr. at 384 (Debbie).

tributed widely to research laboratories across the United States.[309]

309. In September 1976, Andrulonis' antibody titer was measured at 1:8. Exh. 88 (Results of macro serum neutralization tests taken of Griffin personnel between September 21, 1976 and September 23, 1976). Andrulonis received a booster shot of the Lilly vaccine that month, and in October 1976 his antibody titer was measured at 1:32. Tr. at 305–06 (Debbie); Exh. 103 (Jerome Andrulonis' record of immunizations); Exh. 83 (Letter dated December 3, 1977 from Dr. John G. Debbie to Dr. James R. Tillotson). In November 1976, Andrulonis had an antibody titer measured at 1:8 in one test and 1:34 in another. Tr. at 250–51 (Debbie); Exh. 89 (Results of macro serum neutralization tests taken of Griffin personnel between November 1, 1976 and November 3, 1976). On April 21, 1977, after the last Uni-Glatt experiments conducted at Griffin, Andrulonis' antibody titer was measured at 1:8. Exh. 83. All of these antibody titer readings were well above the minimum of 1:5 recommended by the CDC. *See* United States Department of Health, Education, and Welfare, "Lab Safety at the Center for Disease Control," at II–45 (Revised September 1974) (admitted into evidence as Exh. G–136 and hereinafter referred to as "Lab Safety Manual"). In his deposition testimony, Dr. Melvin K. Abelseth indicated that he believed an individual working with the rabies virus should maintain an antibody titer of at least 1:16. Tr. at 626 (Abelseth Deposition). The policy within the rabies laboratory at Griffin (which was under Abelseth's supervision), however, was that a worker exposed to the virus should receive booster shots when his antibody titer dropped below 1:8. *See supra* note 249. Although the Government has argued that Andrulonis had negligently allowed his antibody titer to drop dangerously low, the court finds that the record on the whole simply does not support this contention.

E. *The On-Set of Illness*

On April 14, 1977, approximately two weeks after the last experiment involving the Uni-Glatt machine, Jerome Andrulonis was sent home from work with a headache and a temperature. That night he became violently ill, but by the next morning his condition had improved and he went to work. Over the next five days, Andrulonis would suffer cyclical flu-like symptoms of greater and lesser severity, until finally he decided to visit a physician, who was unable to determine the cause of Andrulonis' illness.[310] Over the subsequent two days, Andrulonis became very listless, having some difficulty walking and becoming somewhat unresponsive to verbal stimuli. By April 20, Andrulonis was unable to dress himself, and his wife Joanna, upon the recommendation of a family doctor, took her husband to the Albany Medical Center Hospital ("AMCH") to consult with a neurologist.[311]

Within a short time after his arrival at the AMCH emergency room, Andrulonis started to act confused and was unable to answer questions posed by hospital personnel.[312] A battery of tests were ordered,

310. Tr. at 1722 (Testimony of Joanna Andrulonis).

311. Tr. at 1722-23 (Joanna Andrulonis).

312. Tr. at 1724 (Joanna Andrulonis).

including a series of CAT scans of the brain, examinations of spinal fluid, tests to measure brain waves (electroencephalograms, or EEGs), and tests to detect heart abnormalities (electrocardiograms, or EKGs).[313] Although the CAT scans taken April 20 were considered normal, Andrulonis' abnormal spinal fluid and changes in consciousness the day he was admitted to AMCH pointed to a form of encephalitis, or inflammation of the brain.[314] The physicians who initially examined Andrulonis quickly concluded that he was most likely suffering from encephalitis caused by a viral infection.[315] Andrulonis was admitted to AMCH following the examination conducted in the hospital's emergency room and was placed in the intensive care unit.[316]

At some point on April 21, the day following his admission to AMCH's intensive care unit, Andrulonis became extremely agitated and hospital personnel were required to use arm restraints to confine him to his bed.[317] Andrulonis' medical records indicate that he suffered delusions, was diso-

313. Tr. at 675–76 (Tillotson).

314. Tr. at 677–78 (Tillotson).

315. *See* Exh. 123 (AMCH Progress Notes).

316. Tr. at 684 (Tillotson).

317. Tr. at 1724 (Joanna Andrulonis).

rientated, and was unable to understand what was said to him.[318] This period of agitation soon passed, however, and Andrulonis became increasingly lethargic and unresponsive to outside stimuli,[319] finally slipping into a coma.[320] Andrulonis was placed on a respirator, was administered various fluids intravenously, and was provided intensive nursing care. He received medications to control a persistent fever and to prevent convulsions.[321]

An EEG taken on April 21 was reported as markedly abnormal with diffuse, slow brain waves, supporting the initial diagnosis of encephalitis. An EEG taken the next day revealed a progression of wave patterns consistent with a viral cause of the encephalitis.[322] Although it was known that Andrulonis had been exposed to the rabies virus in his work, the attending physicians at AMCH did not immediately conclude that Andrulonis had contracted the

318. Exh. 123 (Intensive Care History).

319. Tr. at 685, 694 (Tillotson).

320. Tr. at 1724 (Joanna Andrulonis); Exh. 123 (AMCH Progress Notes).

321. Tr. at 685–86 (Tillotson).

322. Tr. at 705–06 (Tillotson); *see* Exh. 123 (EKG/EMG/EEG test results).

disease of rabies. In his work at Griffin, Andrulonis had been exposed to a number of viruses; moreover, blood tests conducted during Andrulonis' first two days of hospitalization did not reveal an increase in Andrulonis' serum antibodies to rabies.[323] Possibly because of the uncertainty concerning the cause of Andrulonis' illness, no post-exposure treatment with the Lilly vaccine was attempted.[324]

During his first week at AMCH, Andrulonis developed pneumonia.[325] He remained unconscious, and required full respiratory support. On May 3, a tracheostomy was performed, and a metal tube was placed in Andrulonis' throat to assist breathing.[326] Andrulonis suffered significant paralysis, rendering two of his extremities useless.[327] Doctors at AMCH

323. *See* Exh. 123 (AMCH Progress Notes—History Sheet dated April 22, 1977); *see also id.* (Patient histories prepared by Drs. Rockwell and Woodruff dated April 20, 1977).

324. Tr. at 748–51 (Tillotson).

325. Tr. at 1724–25 (Joanna Andrulonis); Exh. 123 (AMCH Progress Notes).

326. Tr. at 681 (Tillotson).

327. Tr. at 695–96 (Tillotson).

continued to attribute Andrulonis' condition to viral encephalitis, cause unknown.[328] Laboratory tests designed to ascertain the etiologic agent that had caused Andrulonis' illness continued.

During the next week, Andrulonis went through the deepest part of his coma before gradually becoming more responsive to stimuli.[329] On May 5, Andrulonis had been unresponsive to even painful stimuli; on May 10, Andrulonis opened his eyes, and by May 17, his spontaneous movements had increased, though he remained "quite rigid" and unconscious.[330] During this phase of his illness, Andrulonis exhibited an erratic pulse and unstable blood pressure, temperature, and respiration, and was administered medication to control this instability. Andrulonis also developed a condition known as myoclonus, a tetanic spasm of the muscles, and was placed on medication intended to control these spasms.[331] On May 27, a gastrostomy was performed, through which a plastic tube was surgically inserted through Andrulonis' abdominal wall and into his stomach. Attempts to

328. Tr. at 686 (Tillotson).

329. Tr. at 688 (Tillotson).

330. Exh. 123 (AMCH Progress Notes); Tr. at 688 (Tillotson).

331. Tr. at 693–94 (Tillotson).

feed Andrulonis through tubes that had been inserted through his nose had not been very successful because of difficulties Andrulonis had had in swallowing. The gastrostomy was performed to avoid this problem by permitting the administration of food directly to the stomach.[332]

Initially, serum neutralization tests did not reveal any noticeable elevation in Andrulonis' rabies antibody titer.[333] On April 26, however, Andrulonis' rabies antibody titer was measured at 1:64, a significant rise.[334] On May 9, Andrulonis' antibody titer was measured at an astronomical 1:65,536; by the end of May, Andrulonis' antibody titer was consistently recorded at 1:200,000.[335] Ultimately, Andrulonis' antibody titer would rise to more than 1:1 million, the highest rabies antibody titer ever recorded in man.[336]

Doctors concluded from the marked increase in Andrulonis' rabies antibody count

332. Tr. at 700–01 (Tillotson); Exh. 123 (AMCH Progress Notes).

333. Tr. at 194 (Debbie); Tr. at 536 (Abelseth Deposition); Exh. 83 (Letter from Debbie to Tillotson).

334. Tr. at 194 (Debbie); Exh. 83.

335. Exh. 83.

336. Tr. at 195 (Debbie).

that the rabies virus had invaded his body and caused his illness.[337] Andrulonis did not receive post-exposure treatment with the Lilly vaccine at this point because his treating and consulting physicians concluded that Andrulonis' antibody titer was so high that administration of the vaccine would have had negligible beneficial effect, while the risks associated with post-exposure vaccination would have remained undiminished.[338] Andrulonis was placed in strict isolation in order to minimize the risk of transmitting the virus to others in the hospital.[339] Studies were then conducted to determine whether a deficiency in Andrulonis' immune system may have contributed to the development of his illness. Those studies showed that Andrulonis had resisted other secondary infections and indicated that his immune system was probably intact when he fell ill.[340]

337. Tr. at 689 (Tillotson).

338. Tr. at 808, 819 (Tillotson).

339. Tr. at 690 (Tillotson).

340. Tr. at 699 (Tillotson).

F. *The Cause of Jerome Andrulonis' Illness*

Although the point is not conceded by the Government or the State, the court finds it manifestly clear that Jerome Andrulonis contracted the disease of rabies, necessitating his hospitalization on April 20, 1977.[341] Andrulonis' antibody titer reached levels vastly exceeding that which could have been achieved by any vaccination program, indicating that Andrulonis' immune system was responding to the presence of rabies virus which had entered his body from some source other than the vaccination regimen to which Andrulonis adhered.[342] Similarly astronomical measures of rabies antibodies in Andrulonis' cerebrospinal fluid evidenced that the rabies virus had invaded his central nervous system.[343]

[1] Moreover, the course of Andrulonis' illness tracked the typical symptomatic progression of the disease of rabies in man as

341. *See* Tr. at 557 (Abelseth Deposition); Tr. at 890 (Winkler Deposition); Tr. at 689, 726 (Tillotson); "Rabies in New York Laboratory Worker," *supra* note 4, at 183.

342. Tr. at 825–27 (Tillotson); Tr. at 557 (Abelseth Deposition); Tr. at 890 (Winkler Deposition); "Rabies in New York Laboratory Worker," *supra* note 4, at 183.

343. *See* Exh. 83.

summarized earlier in this opinion.[344] The first signs of illness Andrulonis exhibited were the flu-like symptoms associated with the prodromal stage of the disease, and these symptoms persisted for five days, as might be expected in a case of rabies exposure.[345] At the time he was hospitalized, Andrulonis was beginning to enter the acute neurologic stage of the disease, and by April 21 Andrulonis was exhibiting the agitation and hyperactivity associated with that stage.[346] Finally, Andrulonis entered the coma stage, although some of the symptoms associated with the acute neurologic stage persisted into the coma stage.[347] The damage to Andrulonis' brain, which is discussed in greater detail below, is consistent with rabies encephalitis.[348]

It is not known with absolute certainty which specific exposure to the rabies virus Jerome Andrulonis experienced caused his illness.[349] However, "[a] plaintiff is not

344. *See* text, *supra* at 1437-1438.

345. *Compare* text, *supra* at 1465, with text, *supra* at 1437.

346. *Compare* text, *supra* at 1465-1466, with text, *supra* at 1437-1438.

347. *Compare* text, *supra* at 1466-1467, with text, *supra* at 1438; *see* Tr. at 697 (Tillotson).

348. *Compare* text, *infra* at 1511-1512, with text, *supra* at 1438.

349. Tr. at 394 (Debbie); Tr. at 890, 915 (Winkler Deposition); Tr. at 743, 804-05 (Tillotson).

obligated to eliminate all possibility that the injuries resulted from causes other than a defendant's negligence." *Ledogar v. Giordano*, 122 A.D.2d 834, 837, 505 N.Y. S.2d 899, 902 (2d Dept.1986). Plaintiffs have demonstrated by a fair preponderance of the evidence that Andrulonis contracted the disease as the result of an aerosol exposure to the ERA–BHK/21 rabies virus strain during the March 29, 1977 Uni-Glatt experiment conducted at Griffin Laboratory in the presence of Drs. Debbie and Baer.[350]

The incubation period of the rabies virus after it has infected a human is variable, lasting from ten days to several months.[351] After it was learned that Andrulonis had contracted rabies, officials from NYSDOH as well as from the CDC investigated the actual or potential exposures to the rabies virus Andrulonis had experienced in the several months preceding April 14, 1977, the day Andrulonis first exhibited symp-

350. Expert opinion testimony supporting this conclusion is found throughout the record. *See, e.g.*, Tr. at 1013 (Hattwick); Tr. at 1264 (Clark); Tr. at 1405–06 (Shope); Tr. at 916 (Winkler Deposition); Tr. at 727–28 (Tillotson); Tr. at 279 (Debbie).

351. See text, *supra* at 1437; Tr. at 970–71 (Tillotson).

toms of the disease.[352] These inquiries failed to uncover any possible exposures to the rabies virus Andrulonis might have had outside of his work.[353] Andrulonis constantly encountered the virus, however, in the course of his employment at Griffin; indeed, all of his work there involved the rabies virus.

In the regular course of his employment, Andrulonis was required to inoculate live animals and to care for live animals which had been exposed to the rabies virus during various research projects. In his diagnostic work, Andrulonis was exposed to specimens taken from animals suspected of having rabies. Andrulonis frequently performed a number of routine laboratory procedures involving the rabies virus, including the preparation of slides for antibody tests, the titration of virus strains used in research projects, the homogenization of virus preparations in a blender, the lyophilization of virus preparations in an apparatus known as a "lyophilizer," and the concentration and purification of rabies virus

352. Tr. at 260–61 (Debbie); Tr. at 558–557A (Abelseth Deposition); Tr. at 885–86 (Winkler Deposition); *see* NYS Retirement System Report, *supra* note 306.

353. Tr. at 728 (Tillotson); "Rabies in New York Laboratory Worker," *supra* note 4, at 183.

through the use of a centrifuge which would spin a tissue culture infected with the virus at high speed.[354] In connection with the Uni-Glatt experiments conducted under the supervision of Dr. Debbie in March 1976, July 1976, September 1976, and March 1977, Andrulonis was responsible for preparing the solutions containing rabies virus strains which were to be sprayed onto the nonpareils suspended within the transparent column of the Uni-Glatt machine. This involved transferring solutions containing virus from vials to flasks and taking samples from those solutions and performing titrations.[355] Finally, Andrulonis was exposed to aerosols containing various rabies virus strains during the Uni-Glatt experiments conducted at the Griffin rabies laboratory in 1976 and 1977.[356]

354. Tr. at 386–93 (Debbie); Tr. at 890–91 (Winkler Deposition); Exh. G-125C at 282–89, 292–93 (Trimarchi Deposition); Exh. G-125A at 248–51 (Rudd Deposition); NYS Retirement System Report, *supra* note 306.

355. *See* Exh. G-125C at 148–49 (Trimarchi Deposition).

356. *See* text, *supra* at 1457–1460 and 1463–1465.

It is not likely that Andrulonis contracted the disease of rabies as a result of an exposure that may have occurred while he carried out his routine daily responsibilities at Griffin. Such exposures regularly occurred in laboratories throughout the United States before March 1977, and none of the individuals exposed—many of whom had immunization histories and work exposure histories substantially identical to that of Andrulonis—developed the rabies disease.[357] The experience of rabies researchers indicated that the Lilly vaccine would offer protection against an exposure to the virus through a bite from an infected animal, accidental self-inoculation, or exposure of a cut or skin abrasion to the virus.[358] Further, the body's own defense mechanisms were probably adequate to defend against the mild aerosol exposures that occasionally occurred during routine laboratory procedures.[359]

The court's conclusion that Andrulonis did not develop disease as a result of an exposure while he performed routine laboratory procedures is bolstered by the evidence offered concerning Andrulonis' work habits. Andrulonis was considered by his

357. Tr. at 1014 (Hattwick).

358. Tr. at 1029-30, 1059-60 (Hattwick); Tr. at 728 (Tillotson).

359. Tr. at 1101 (Hattwick).

superiors and co-workers to be diligent and conscientious in completing the tasks assigned to him. He was a careful worker, adept at performing tasks that required finesse, and was safety conscious in carrying out his work.[360] There was an unwritten policy at Griffin requiring workers to report laboratory accidents or unintended exposures to virus, and Andrulonis had not reported any such accident or exposure.[361] When all of the factors are considered together, it is unlikely that Andrulonis was accidentally infected by rabies virus while performing the routine laboratory tasks for which he was responsible. Andrulonis' illness was more likely the result of an "unusual" exposure.[362]

The exposure Andrulonis had to the virus when the Uni-Glatt machine was in operation during the four enteric coating experiments conducted at Griffin was certainly "unusual."[363] The aerosols containing rabies virus that were created during the

360. Tr. at 313–15 (Debbie); Tr. at 608–09 (Abelseth Deposition).

361. Tr. at 609 (Abelseth Deposition); "Rabies in New York Laboratory Worker," *supra* note 4, at 183 (no known laboratory exposures other than during Uni-Glatt experiments).

362. Tr. at 1013–15 (Hattwick); Tr. at 728–29 (Tillotson).

363. *See* Tr. at 1063–64 (Hattwick).

Uni-Glatt experiments were not contained. The Uni-Glatt machine leaked heavily, a large quantity of virus was aerosolized in each experiment and Andrulonis worked in close proximity to the machine for long periods of time during each experiment. Whatever exposures Andrulonis had to aerosolized virus while performing routine laboratory procedures—and the evidence is inconclusive that such exposures occurred at all [364]—were dwarfed by the magnitude of

364. *See* "Rabies in New York Laboratory Worker," *supra* note 4 at 183 (other than Uni-Glatt experiments, "[n]o accidental exposure to rabies virus is known or suspected to have occurred in the laboratory . . "). Aerosols containing rabies virus can be produced when a virus strain is homogenized in a blender, but the evidence indicates that at Griffin this procedure was routinely conducted within a physical containment system that would prevent exposure of the worker performing this task to aerosols. NYS Retirement System Report, *supra* note 306; Tr. at 266 (Debbie). Virus can also be aerosolized through a procedure known as centrifugation. The Griffin laboratory had three centrifuges, one of which was airtight. Tr. at 388 (Debbie). Nothing in the record indicates that the centrifuges that were not airtight were used by Andrulonis in purifying virus strains. Finally, it appears that if a procedure known as lyophilization is improperly performed, aerosols can be produced. *See* Tr. at 1096 (Hattwick). There is no evidence that Andrulonis was exposed to aerosols when he performed this procedure.

the exposures that occurred during the Uni-Glatt experiments, and it was during these experiments that Andrulonis more likely than not came into contact with the virus strain that caused his illness.[365]

Given the variable incubation period associated with the rabies virus, it is conceivable that any of the exposures that occurred during the various Uni-Glatt experiments conducted at Griffin between March 1976 and March 1977 could have resulted in Andrulonis' infection and disease. Nonetheless, the evidence that the exposure that caused his illness occurred during one of the two coating runs conducted in March 1977 is compelling. The other Uni-Glatt experiments were conducted six or more months before Andrulonis exhibited the first symptoms of this illness, and an incubation period of such a length, though theoretically possible, is well outside the average range of twenty to sixty days.[366] Only fourteen percent of all cases have incubation periods exceeding ninety days, regardless of route of exposure or the proximity

365. *See* Tr. at 1050–51, 1094–95 (Hattwick) (The aerosol exposures Andrulonis experienced during the Uni-Glatt experiments were "not at all comparable" to other exposures that may have occurred in the laboratory, and had such exposures not occurred, Andrulonis would not have contracted the disease of rabies).

366. *See* text, *supra* at 1437.

of that exposure to the central nervous system.[367] Because an aerosol exposure was likely the cause of Andrulonis' illness and because an individual's olfactory nerves are but a short distance from the brain, a short incubation period was more probable than a long one.[368] The aerosol exposures Andrulonis experienced during the Uni-Glatt experiments were intensive, and this factor also argues for a short incubation period rather than a long one.[369] In short, the timing of the onset of Andrulonis' illness indicates that he was exposed to the virus strain that caused his illness during the Uni-Glatt experiments of March 1977.[370]

There is one factor that not only further supports the conclusion that Andrulonis' disease was the result of an exposure in March 1977 but also establishes that it is likely that the exposure occurred during the use of the ERA-BHK/21 virus strain Baer had prepared. That factor is the extremely high titer of the strain supplied by

367. "The Disease in Man," *supra* note 12, at 289.

368. "The Disease in Man," *supra* note 12, at 289 (short incubation period likely if exposure site is close to head).

369. Tr. at 970–71 (Hattwick); *see* text, *supra*, at 1459.

370. Tr. at 1015, 1084, 1098 (Hattwick); Tr. at 279 (Debbie).

Baer. The titer of the ERA-BHK/21 virus strain used in the March 29 Uni-Glatt experiment was 8.1; the titers of the other virus strains used in the Uni-Glatt did not exceed 6, and the titer of the ERA-SMB strain used in the Uni-Glatt machine on March 30, 1977 was 5.5.[371] Thus, the ERA-BHK/21 virus strain contained over one hundred times as many rabies viral particles per given volume than any other virus strain used in the machine, and was almost 400 times as concentrated at the ERA-SMB strain used March 30, 1977. The discrepancy in the intensity of the exposure to airborne virus during the March 29 experiment and the other Uni-Glatt experiments may have been magnified by the fact that the heat generated by the Uni-Glatt machine while it was in operation had been killing most of the viral particles in the virus strains previously used in Uni-Glatt experiments.[372] The ERA-BHK/21 strain is more resistent to heat than other strains, such as the ERA-SMB, which were usually used in the previous Uni-Glatt experiments. Further, the virus strain used in the March 29 experiment had been passaged and concentrated to an extremely high titer for the very purpose of assuring

371. *See* text, *supra*, at 1459–1460, 1462–1463 and 1464–1465.

372. *See* text, *supra* at 1460.

that a significant number of viral particles would survive the heat of the machine during the coating process. As a consequence of all of these factors, Andrulonis' exposure to rabies viral particles during the March 29 coating run was markedly greater than during the runs conducted with the other virus strains.

As noted above, the quantity of viral particles to which an animal is exposed is the most important variable governing the chances that rabies virus will infect the animal after an airborne exposure;[373] Andrulonis' exposure to aerosolized viral particles during the experiment in which the ERA-BHK/21 virus strain with a titer of 8.1 was used could fairly be characterized as "massive."[374] The extraordinary difference in the magnitude of the exposure to aerosolized virus Andrulonis experienced on March 29, 1977 and the exposure experienced during the other coating runs was a critical consideration that led those experts who testified about the cause of Andrulonis' illness to conclude that it was the March

373. *See* text, *supra* at 1447-1448.

374. Exh. G-125H at 210-11 (Huffaker Deposition, February 2, 1983); *see also* Tr. at 1062-64 (Hattwick).

29 exposure that most likely resulted in infection and disease.[375]

In sum, the court accepts the opinion testimony of the various experts that it could be said with a reasonable degree of medical and scientific certainty that Andrulonis contracted the disease of rabies as a result of his exposure to the aerosolized ERA-BHK/21 virus strain used in the March 29 Uni-Glatt experiment.[376] Plaintiffs have established by a fair preponderance of the evidence that the likelihood that the injuries suffered by Jerome Andrulonis were caused by his exposure to aerosols containing the ERA-BHK/21 virus strain that escaped from the Uni-Glatt machine on March 29 far outweighs the combined prospects that his injuries were caused by some other exposure, either potential or actual, either known or unknown.

375. *See, e.g.*, Tr. at 1062–64 (Hattwick); Tr. at 1431, 1441 (Shope); Tr. at 728 (Tillotson); Tr. at 279 (Debbie).

376. See *supra* note 350.

IV. LIABILITY

[2] The only claims made by plaintiffs that remain unresolved in this case are those of plaintiff Jerome Andrulonis against the United States under the Federal Tort Claims Act. By enacting the FTCA, Congress waived the Government's sovereign immunity in a broad class of cases by permitting a private person to maintain a suit for damages against the United States

> for injury or loss of property, or personal injury or death caused by the negligent or wrongful act or omission of any employee of the Government while acting within the scope of his office or employment, under circumstances where the United States, if a private person, would be liable to the claimant in accordance with the law of the place where the act or omission occurred.

28 U.S.C. § 1346(b); *see also id.* § 2674. The court concludes that the substantive law of New York is applicable in the present case because most of the acts or omissions on the part of Government employees that had "the most significant causal effect" in reference to the injuries suffered by Jerome Andrulonis occurred in Albany, New York. *See Bowen v. United*

States, 570 F.2d 1311, 1318 (7th Cir. 1978).[377]

377. Plaintiffs' primary claims are based on the adequacy of the warnings given by Dr. Baer when he supplied the ERA-BHK/21 virus strain to Debbie on March 28, 1977 and when he witnessed the Uni-Glatt experiment conducted the following day as well as Baer's failure to intervene and reclaim the virus strain he prepared when he should have realized the March 29 experiment was being conducted unsafely.

These "act[s] or omission[s]" occurred in Albany. Not all of plaintiffs' allegations of negligence involve acts committed in New York, however. Plaintiffs have argued that Dr. Baer was negligent in passaging and concentrating the ERA-BHK/21 virus strain used in the March 29 Uni-Glatt experiment to a high titer and by failing to "test" that virus strain before supplying it to Debbie. These alleged acts or omissions occurred in Georgia. Plaintiffs also allege that the Government is liable for harm to Andrulonis that resulted from negligent misrepresentations made in the letters prepared by Drs. Baer and Debbie and submitted to representatives of WARF. *See* text, *supra* at 1455-1456. Presumably, these letters were written in Georgia.

The FTCA imposes liability on the Government "in accordance with the law of the place where the act or omission occurred." 28 U.S.C. § 1346(b). The Supreme Court has held that the "law of the place" to be applied includes the choice-of-law rules of the state in which the act or omission occurred. *Richards v. United States*, 369 U.S. 1, 82 S.Ct. 585, 7 L.Ed.2d 492 (1962). Under Georgia law, when an injury suffered in one state is caused by negligent conduct in another state, the law of the place where the injury occurred governs and controls a plaintiff's cause of action. *See Ellington v.*

Because the only unresolved claims by Jerome Andrulonis are against the Government, if the court does not find that an employee of the United States breached a duty owed Andrulonis and that that negligence was a substantial cause of the event which produced his injuries, *see Derdiarian v. Felix Contracting Corp.*, 51 N.Y.2d 308, 315, 434 N.Y.S.2d 166, 169, 414 N.E.2d 666, 670 (1980), the culpable conduct of the other parties involved in this case, including that of employees of the State of New York, would be irrelevant. Nonetheless, it is easier to understand Andrulonis' claims against the Government if the negligent conduct imputable to NYSDOH is reviewed first. Accordingly, the court will examine the basis for the Government's third-party claim for contribution against the State before turning to Andrulonis' claims against the United States.

Tolar Const. Co., 142 Ga.App. 218, 235 S.E.2d 729 (3d Div.), *cert. dismissed*, 239 Ga. 849, 240 S.E.2d 551 (1977). Thus, for those theories of recovery in which the allegedly negligent conduct of Government employees took place in Georgia, New York law applies.

A. *The Culpable Conduct Attributable to NYSDOH*

[3] When the Government is sued under the FTCA, it may commence a third-party action against another party seeking contribution, provided that such an action is allowed under local law. *United States v. Yellow Cab Co.*, 340 U.S. 543, 551–52, 71 S.Ct. 399, 405, 95 L.Ed. 523 (1951). In the case at bar, New York law governs whether the Government can assert a contribution claim. *Compare Barrett v. United States*, 853 F.2d 124, 128–29 (2d Cir.1988), *cert. denied*, — U.S. ——, 109 S.Ct. 866, 102 L.Ed.2d 990 (1989); *Barron v. United States*, 654 F.2d 644, 647–48 (9th Cir.1981).

[4–7] Under New York law, NYSDOH is insulated from suit by plaintiff Jerome Andrulonis because his injuries resulted from an accident that occurred in the course of his employment with the State. N.Y.Work.Comp.Law §§ 11, 29(6) (McKinney Supp.1989). Notwithstanding the provisions of its Workers' Compensation Law, however, a defendant alleged to be responsible for a plaintiff's injuries may bring a third-party action against the plaintiff's employer if the employer's negligence was also a proximate cause of the injuries suffered. *Dole v. Dow Chemical Co.*, 30 N.Y.2d 143, 153, 331 N.Y.S.2d 382, 391, 282 N.E.2d 288, 294 (1972). The State of New York has waived its immunity from suit in tort, *see* N.Y.Ct.Cl.Act § 8 (McKinney

1963), and a joint tortfeasor's claim for contribution under N.Y.C.P.L.R. §§ 1401-1404 may be maintained against the State. *See Bay Ridge Air Rights, Inc. v. State*, 44 N.Y.2d 49, 404 N.Y.S.2d 73, 375 N.E.2d 29 (1978). When the United States has been sued under the Federal Tort Claims Act, it may bring a third-party action for contribution against the State of New York in federal district court without offending the eleventh amendment of the Constitution. *Barrett v. United States*, 853 F.2d 124; *compare United States v. Hawaii*, 832 F.2d 1116 (9th Cir.1987).

[8–10] In New York, "[a] claim for contribution exists only when two or more tort-feasors share in responsibility for an injury in violation of duties they respectively owed to the injured person." *Smith v. Sapienza*, 52 N.Y.2d 82, 87, 436 N.Y.S.2d 236, 238, 417 N.E.2d 530, 532 (1981); *New York v. City of Johnstown*, 701 F.Supp. 33, 37 (N.D.N.Y.1988) (applying New York law). Thus, to recover on its third-party claim in the event it is found liable to plaintiff Jerome Andrulonis under the FTCA, the Government must demonstrate that NYSDOH or its agents had a duty recognized by the law that required them to conform to a certain standard of conduct in order to avoid exposing Andrulonis to unreasonable risks, that the duty owed was breached, and that the injury suffered was a direct and proximate result of that breach. *See Akin v. Glens Falls City*

School District, 53 N.Y.2d 325, 333, 441 N.Y.S.2d 644, 648, 424 N.E.2d 531, 535 (1981); *Caraballo v. United States*, 830 F.2d 19, 21 (2d Cir.1987) (applying New York law); W. Prosser & W. Keeton, *The Law of Torts* § 30, at 164–65 (5th ed. 1984) (hereinafter Prosser & Keeton).[378]

378. Because the Government's third-party claim for contribution is governed by local law, the Government can assert a claim for contribution claim only if such a claim would be available to a private party under the same circumstances in New York. The State argues that the Government's third-party claim is precluded by the "discretionary conduct" immunity it enjoys under New York law. *See Arteaga v. State*, 72 N.Y.2d 212, 216–17, 532 N.Y.S.2d 57, 59, 527 N.E.2d 1194, 1196 (1988); *Tango v. Tulevech*, 61 N.Y.2d 34, 40, 471 N.Y.S.2d 73, 76, 459 N.E.2d 182, 185 (1983). While the State, by enacting N.Y.Ct.Cl. Act § 8, "assum[ed] liability under the rules applicable to corporations and individuals for the actions of its officers and employees in the everyday operations of government, . . . [it] retained its immunity for those governmental actions requiring expert judgment or the exercise of discretion." *Arteaga*, 72 N.Y.2d at 216, 532 N.Y.S.2d at 59, 527 N.E.2d at 1196 (citations omitted). Under this rule, absolute immunity is conveyed upon the State and its agencies when the action of one of its employees "involves the conscious exercise of discretion of a judicial or quasi-judicial nature." *Id.* at 216, 532 N.Y.S.2d at 59, 527 N.E.2d at 1196; *see Tarter v. State*, 68 N.Y.2d 511, 510 N.Y.S.2d 528, 503 N.E.2d 84 (1986). A discretionary act is deemed "quasi-judicial" under New York law if it entails "discretionary decisions in furtherance of general policies and purposes where the exercise of reasoned judgments can produce dif-

[11] An employer such as NYSDOH has a duty to exercise reasonable care in assuring that its employees are provided a safe work environment and that equipment used in the performance of a work task is safe and appropriate for that task. *Simone v. Kirk*, 173 N.Y. 7, 13, 65 N.E. 739 (1902); *McGuire v. Bell Telephone Co.*, 167 N.Y. 208, 211–12, 60 N.E. 433 (1901); *Byrne v. Eastmans Co.*, 163 N.Y. 461, 465, 57 N.E. 738 (1900); Prosser & Keeton,

ferent acceptable results." *Arteaga*, 72 N.Y.2d at 219, 532 N.Y.S.2d at 61, 527 N.E.2d at 1198. Dr. Debbie's decision to pursue research aimed at developing a means of immunizing wildlife against rabies might fall within this definition, and arguably so would his decision to attempt to develop enterically coated bait that could be distributed in the wild. The *manner* in which these decisions were implemented, however, is not shielded by "discretionary conduct" immunity. *Compare* text, *infra* at 1496–1498 (discussing the immunity of the United States under the discretionary function exception of 28 U.S.C. § 2680(a)). Once a choice between competing alternatives that "can produce different acceptable results" has been made, liability may result from a failure to effectuate that choice in a non-negligent manner. *Compare Friedman v. State*, 67 N.Y.2d 271, 286–88, 502 N.Y.S.2d 669, 676–77, 493 N.E.2d 893, 900–01 (1986). The Government's third-party claim against NYSDOH is predicated on allegations that Debbie failed to carry out the discretionary policy decision he made to pursue the enteric coating process in a non-negligent manner, and thus NYSDOH is not entitled to immunity from that claim.

§ 80, at 569. In the case at bar, Dr. Debbie, the scientist in charge of supervising Griffin's rabies laboratory, was charged with the responsibility of assuring that the rabies laboratory was reasonably safe for employees who worked there.[379] In assessing whether that duty has been breached, the court must determine "whether the resulting injury was a reasonably foreseeable consequence" of the conduct of the employer or its agents. " 'If the defendant could not reasonably foresee any injury as the result of his act, or if his conduct was reasonable in the light of what he could anticipate, there is no negligence, and no liability.' " *Danielenko v. Kinney Rent A Car, Inc.*, 57 N.Y.2d 198, 204, 455 N.Y.S.2d 555, 557, 441 N.E.2d 1073, 1075 (1982) (quotation omitted); *see also Gordon v. City of New York*, 70 N.Y.2d 839, 841, 523 N.Y.S.2d 445, 446, 517 N.E.2d 1331, 1332 (1987).

[12, 13] When a defendant possesses or purports to possess specialized knowledge and his conduct falls within the ambit of that knowledge, a court's inquiry into whether an event or result was reasonably foreseeable to that defendant must take

379. Tr. at 385 (Debbie); Exh. G-125H (Huffaker Deposition, February 1, 1983); *see* Tr. at 1306–07 (Clark) (discussing duty of supervising scientist to assure the safety of those working under him).

into account that specialized level of learning. *Compare Cornbrooks v. Terminal Barber Shops, Inc.*, 282 N.Y. 217, 222, 26 N.E.2d 25 (1940) (barbers); *Allan v. The State Steamship Co.*, 132 N.Y. 91, 95, 30 N.E. 482 (1892) (druggists). Professional persons "are required not only to exercise reasonable care in what they do, but also to possess a standard minimum of special knowledge and ability." Prosser & Keeton § 32, at 185. In this respect, a scientist who engages in the sort of research Dr. Debbie directed at Griffin should be held to a standard of knowledge and ability analogous to that imposed on a physician specializing in a certain area of medicine. The law places upon one practicing as a physician or surgeon

> the duty of possessing, that reasonable degree of learning and skill that is ordinarily possessed by physicians and surgeons in the locality where he practices, and which is ordinarily regarded by those conversant with the employment as necessary to qualify him to engage in the business of practicing medicine and surgery. Upon consenting to treat a patient, it becomes his duty to use reasonable care and diligence in the exercise of his skill and the application of his learning to accomplish the purpose for which he was employed.... The law holds him liable for an injury to his patient resulting from want of the requisite knowl-

edge and skill, or the omission to exercise reasonable care....

Pike v. Honsinger, 155 N.Y. 201, 209, 49 N.E. 760 (1898); *see also Littlejohn v. State*, 87 A.D.2d 951, 952, 451 N.Y.S.2d 225, 226 (3d Dept.1982); *Twitchell v. MacKay*, 78 A.D.2d 125, 127–28, 434 N.Y.S.2d 516, 518 (4th Dept.1980). Similarly, in undertaking the research involving the Uni-Glatt machine, Dr. Debbie assumed responsibilities that could only be discharged by those possessing the "reasonable degree of learning and skill" deemed necessary to engage in sophisticated rabies research by those familiar with the experimental use of the rabies virus.

[14] The court finds that in conducting the March 29, 1977 Uni-Glatt experiment, Debbie was engaged in research that required the expertise of someone who was a member of the "community of rabies experts," [380] defined at trial to include twenty-

380. It is the responsibility of any researcher supervising experiments in which live rabies virus strains are used to become knowledgeable of the potential consequences of human exposure to those strains. Tr. at 1288–89 (Clark). Moreover, as head of Griffin's rabies laboratory, Debbie was responsible for keeping abreast of new developments in the rabies field, Tr. at 458 (Debbie), and with regard to the March 29, 1977 Uni-Glatt experiment, it was Debbie's responsibility to become familiar with the literature pertaining to the ERA-BHK/21 virus strain. Tr. at 1290 (Clark).

five to fifty scientists nationwide.[381] Because this is an elite group with limited numbers, the relevant "locality" in this case would be the United States as a whole. In essence, this core group of rabies experts largely "establish [their] own standard of care," *Toth v. Community Hospital*, 22 N.Y.2d 255, 262, 292 N.Y.S.2d 440, 447, 239 N.E.2d 368, 372 (1968), since the factual evidence introduced at trial through these experts to a great degree defines the minimum level of knowledge required of someone engaged in sophisticated research involving the rabies virus.

To summarize what the court has discussed in detail above, in March 1977 a member of the community of rabies experts, to safely and effectively engage in rabies research, should have understood the basic principle that passage of a rabies virus strain in any host system can result in changes in the biological properties and characteristics of the virus strain pas-

381. *See supra* note 7.

saged.[382] Passaging can lead to mutations, and such mutations can be particularly unpredictable when—as was the case with the ERA-BHK/21 virus strain—the virus has been passaged in more than one cell system.[383] It was known that virus strains adapted to tissue culture tended to have a greater ability to cause disease after inhalation. A researcher using a virus strain derived from the ERA commercial vaccine in 1976 and 1977 should have been aware that it was not known whether such a strain could cause disease in man. It should have been known that derivatives of

382. *See* Tr. at 1276–77 (Clark); Tr. at 1425–26 (Shope); *see also* text, *supra* at 1439–1441. At trial, the Government objected on various grounds to a series of questions addressed to Dr. H Fred Clark, seeking his opinion regarding what "Dr. George Baer knew or should have known," with the State occasionally joining on the objections. *See* Tr. at 1272–81. The court reserved decision on all of these objections. Obviously, Dr. Clark has no personal knowledge of what Dr. Baer knew, and the court disregards this aspect of the challenged testimony. With regard to what Baer "should have known," Clark had testified that Baer was a member of the community of rabies experts that has been defined in this trial, *see* Tr. at 1251, and indeed most of the challenged questions were couched in terms of the knowledge of "the rabies community, the scientific community of research scientists dealing with rabies." The court will consider Clark's testimony to the extent that he discussed what should have been known within the community of rabies experts.

383. *See* Tr. at 1451 (Shope); *see* text, *supra* at 1461.

the CRA vaccine usually had "high specific infectivity." Moreover, it should have been known that the ERA-BHK/21 virus strain had proven to be pathogenic to a number of species of rodents, with unpredictable differences in pathogenicity for closely related species.[384] Absent reliable data concerning the pathogenicity of the ERA-BHK/21 strain, safe laboratory practice required an assumption that the strain was virulent to humans.[385]

Further, a member of the community of rabies experts in 1976 and 1977 should

384. *See* text, *supra* at 1461-1462.

385. Tr. at 2012-13, 2021-22 (Richardson); Tr. at 1452 (Shope); *see* "MMWR on Rabies Immunization," *supra* note 278, at 406 ("There is no reliable information on which to judge the risk associated with accidental human exposure to new animal vaccines incorporating [the ERA] strain[ ] in other substrates or to animal vaccines incorporating other rabies virus strains, and they should be regarded as potentially virulent for purposes of managing the treatment of exposed humans."); "Follow-up on Rabies," *supra* note 4, at 249 ("The pathogenicity of attenuated vaccine strains varies with the site of inoculation, strain of virus, and species exposed. Attenuated strains which have been further manipulated, as by tissue culture or animal passage, are of unknown virulence and must be considered pathogenic until proven otherwise.").

have known that as a general rule the concentration of a particular virus strain directly affects its pathogenicity, especially when an aerosol exposure occurs.[386] It was common knowledge among rabies researchers that a virus strain with a comparatively high titer ordinarily contains a high concentration of viral particles, and that a strain with a titer as high as that of the ERA-BHK/21 strain used in the March 29 Uni-Glatt experiment would be extraordinarily more concentrated than the ERA commercial vaccine, or even the strains previously used in Uni-Glatt experiments.[387]

[15] Someone performing experiments involving the rabies virus in which aerosolized virus is produced must also be cognizant of the unique hazards posed by the creation of such aerosols. At least three human deaths had been attributed to aerosol exposures to the rabies virus before 1977, and thus it was known within the community of rabies experts that humans were susceptible to contracting the disease

386. *See* text, *supra* at 1447–1448; Tr. at 1451–52 (Shope).

387. *See* text, *supra* at 1470–1471.

through the airborne route.[388] Further,

388. The question of precisely *how* susceptible man is to infection by the aerosol route was strenuously contested at trial. By March 1977, only three human deaths had been linked to the airborne transmission of the rabies virus, a minuscule fraction of the total number of deaths that have been attributed to the rabies virus in human history. It seems, however, that humans rarely encounter natural environments where aerosols containing a substantial amount of rabies virus are present. The research and observations made by Constantine, *see* text, *supra* at 1445–1447, suggested the possibility that humans, who are highly resistent to developing the disease of rabies as a result of animal bite exposures, may be more susceptible to the disease when exposed to aerosolized virus than foxes and coyotes, species known to be particularly susceptible to the disease after infection through conventional routes. *See* Constantine Report, *supra* note 102, at 26. On the other hand, none of the many investigators exposed to the atmosphere of Frio Cave for significant periods of time in the 1960s while studying the airborne transmission of the virus contracted rabies, suggesting that man is very resistent to the disease even when exposed to airborne virus. *See* Tr. 2247–52 (Baer) (describing minimal precautions taken by researchers who conducted Frio Cave experiments in the 1960s). Dr. Winkler concluded in his report on the laboratory worker who contracted rabies after being exposed to aerosolized virus while using a blender that "the inhalation route ... seems to be one of low infectivity." "Laboratory Worker," *supra* note 120, at 1221.

The court does not assign much significance to this issue, despite the substantial proof offered concerning it. What seems clear is that in March 1977 it was not known with any certainty how vulnerable man was to acquiring the dis-

someone conducting research in which rabies virus was aerosolized should have been aware that whether disease will result from an aerosol exposure to the virus is closely linked to the duration and intensity of the exposure. Finally, that researcher should have been aware of the studies concerning the pathogenesis of an aerosol infection indicating that it was possible that the Lilly vaccine might not offer any protection in the event of infection by the airborne route.

Dr. Debbie knew that the Uni-Glatt machine used during the March 29, 1977 experiment was not airtight and that the machine expelled aerosols into the laboratory atmosphere,[389] and he should have been aware that Jerome Andrulonis was exposed to those aerosols. A member of the community of rabies experts should have known that the virus prepared by Dr. Baer for the March 29 experiment was potentially pathogenic, and that an aerosol contain-

ease of rabies as a result of an aerosol exposure to the virus. In cases where the disease that could potentially result is as serious as rabies, safe laboratory practice required the assumption that man is susceptible to the disease through the threatened route of exposure unless there is clear evidence to the contrary, and the precautions taken must account for this assumption. *See* Tr. at 1452 (Shope) (where knowledge is incomplete, researcher must assume a "worst-case scenario").

389. *See* text, *supra* at 1458–1459.

ing that virus might present a significant risk to those exposed to it.[390] Debbie knew that a human can contract the disease of rabies as a result of an aerosol exposure,[391] should have known that the concentration of virus to which an individual is exposed is likely the most important factor in determining the probability that disease will result from that exposure, and should have realized that the concentration of the ERA-BHK/21 virus strain prepared by Dr. Baer was unusually high. Moreover, he should have recognized that because Andrulonis worked in close proximity to the Uni-Glatt machine for substantial periods of time while a highly concentrated virus strain was being aerosolized within the machine, Andrulonis was being exposed to a massive dose of viral particles. Finally, Debbie should have known that the effectiveness of the Lilly vaccine in preventing disease in the event of such exposure was uncertain.

These facts define the hazards posed to laboratory workers by the Uni-Glatt experiments that Debbie should have been able to anticipate. To determine whether the precautions taken by Debbie were commensurate with the risk he should have per-

390. Tr. at 1274–75 (Clark); *see also* Tr. at 1427 (Shope). The objections to Dr. Clark's testimony are overruled.

391. Tr. at 256–59 (Debbie).

ceived—that is, whether Debbie's conduct was "reasonable"—the court must look to the standards and principles of laboratory safety that prevailed in the scientific community in 1977.

In the 1940s and 1950s, the specialized study of biological safety in the laboratory setting arose out of an increased awareness of the numerous incidents of laboratory-acquired illnesses among researchers and diagnosticians exposed to biological microorganisms.[392] As a result of the work of specialists in biological safety, certain basic principles evolved concerning the methodological evaluation of the hazards associated with a particular laboratory activity.[393] These principles of "risk assessment" helped define what was considered reasonably safe laboratory practice in March 1977, and any investigator conducting laboratory research with hazardous microorganisms was obliged to apply these principles to the task he was undertaking and assess the risk to which those working under the researcher was exposed.[394]

392. Tr. at 1455–57 (Testimony of Dr. Daniel F. Liberman).

393. Tr. at 1457–59 (Liberman).

394. *See* Tr. at 1289 (Clark).

In 1977, proper risk assessment required consideration first of what was known and not known about the organism used.[395] It was important to know whether the organism was a pathogen capable of causing disease and, if so, what concentration of the organism was ordinarily necessary to cause disease, whether the organism could produce illness in humans, the severity of the illness the organism could cause, and the routes of infection through which the organism could invade a host animal.[396] Next, the researcher was required to determine whether the procedures and practices within the laboratory were adequate given the nature and characteristics of that organism, with emphasis being placed on the familiarity of laboratory workers with clearly defined procedures designed to minimize exposures.[397] Finally, proper risk assessment required inquiry into whether the equipment and facilities used for a particular laboratory activity were adequate, an inquiry that usually turned on whether the level of containment provided for an etiologic agent was commensurate with the

395. Tr. at 1459 (Liberman).

396. Tr. at 1459–61, 1462–63 (Liberman).

397. Tr. at 1463–64 (Liberman).

level of hazard that reasonably should have been perceived for a given work activity.[398]

398. Tr. at 1465 (Liberman). A great deal of evidence was presented at trial establishing that the ventilation system in the rabies laboratory failed to meet the minimum standards of safety given the sort of work undertaken in those experiments. The laboratory was heated and cooled by wall units which obtained air from the outside, Tr. at 542 (Abelseth Deposition); Exh. G-125H at 19 (Huffaker Deposition, February 16, 1983), and was ventilated by a fan from an adjacent room that forced air into the laboratory through an open door. Tr. at 172-73 (Debbie); *see* Mallison Report, *supra* note 221, at 1. The laboratory had no forced-air exhaust, and thus forcing air into the laboratory in this manner placed the laboratory under strong positive pressure. *See* Mallison Report, *supra*, at 1; Tr. at 542 (Abelseth Deposition). Air contaminated with hazardous agents—the rabies virus strains used in the various Uni-Glatt experiments—was forced into uncontaminated areas, such as Griffin's corridors. Exh. G-125H at 19-20 (Huffaker Deposition, February 16, 1983). In March 1977 safe laboratory practice required that if an etiologic agent would be suspended in aerosols during the course of an experiment, the room in which the experiment was conducted must be kept under negative air pressure so that individuals outside of the main work area would not be exposed to the aerosolized etiologic agent. *See* Tr. at 2039-40 (Richardson); Tr. at 1332 (Clark). This practice did not have a significant effect on the safety of individuals within the main work area in which aerosols were created. Because the inadequate ventilation of the rabies laboratory at Griffin did not increase the hazard encountered by Andrulonis, the court relegates its discussion of this issue to this footnote.

In assessing the risk of a particular laboratory procedure, importance was placed on whether the etiologic agent used could infect humans if inhaled,[399] since aerosols constituted the route of infection in perhaps a majority of laboratory accidents that occurred through the mid-1970s.[400]

399. *See* Tr. at 1462 (Liberman); Department of Health, Education and Welfare, "Classification of Etiologic Agents on the Basis of Hazard" at 1 (admitted into evidence as Exh. 114 and hereinafter referred to as "Classification of Etiologic Agents") ("Aerosol studies ... increase the hazard" associated with use of etiologic agents in the laboratory).

400. Of the nearly 4,000 cases of laboratory-acquired illnesses that had been studied by the mid-1970s, approximately half were directly ascribed to the creation of aerosols in the laboratory, and the actual percentage of illnesses resulting from aerosol exposures may have been greater than that. Approximately twenty percent of the cases studied were attributable to specific causes such as the failure of laboratory equipment, self-inflicted cuts or injections, or an improper technique for siphoning solution from one flask or test tube to another called "mouth pippetting." In most of the remaining cases in which illnesses were not directly attributed to aerosol exposures, the cause of the illnesses were not determined. Tr. at 1461-62 (Liberman); *see also* Tr. at 2197 (Richardson).

As a consequence, it was widely considered safe laboratory practice to avoid the production of aerosols in the laboratory altogether if possible to do so without undermining the purpose of a particular study, and to contain aerosols if they are necessarily produced in the course of a particular laboratory procedure.[401]

[16] Applying risk assessment principles to the March 29, 1977 Uni-Glatt experiment in which the ERA-BHK/21 rabies virus strain provided by Dr. Baer was used, the court finds that the safety precautions taken in conducting this experiment failed to meet the minimum standards of laboratory safety that then prevailed in the scientific community in general and in the community of rabies experts in particular.[402] As noted above, reasonably safe laboratory practice required the assump-

401. Exh. G-125H at 32 (Huffaker Deposition, February 1, 1983).

402. *See* Tr. at 1016 (Hattwick) (expert opinion); *see also* Tr. at 1479-83 (Liberman) (expert opinion regarding risk assessment). Although Dr. Liberman was not qualified as an expert on the rabies virus, his opinion regarding whether the March 29, 1977 experiment was conducted in conformance with good laboratory practice was based on assumptions supported by the record. The court finds that Dr. Liberman's lack of specialized knowledge of the rabies virus did not disqualify him as an expert on biological safety, and the court receives his testimony over the Government's objections.

tion that the virus strain prepared by Baer was pathogenic. It was known that the disease caused by the rabies virus was extremely severe, resulting in death in virtually all cases in which the disease develops. It was known in March 1977 that man is susceptible to contracting the disease of rabies after an aerosol exposure to the rabies virus, and that the odds of being infected through the airborne route of transmission increased as the amount of viral particles in the aerosol to which one is exposed increased. It was not known whether serum antibodies created by rabies vaccines such as DEV would provide protection to an individual so exposed. In March 1977, it was established within the relevant community of rabies experts that it was not good, safe laboratory practice to expose laboratory workers to an aerosolized rabies virus that had a titer as high as the ERA-BHK/21 virus strain used in the March 29 experiment.[403]

The workers at Griffin involved in the Uni-Glatt experiments knew that aerosols containing rabies virus were produced when the machine was in operation, that the Uni-Glatt was not airtight, and that when the ERA-BHK/21 virus strain provided by Baer was used, an unusually concentrated virus strain was being aerosol-

403. Tr. at 1016 (Hattwick); Tr. at 1281-82 (Clark).

ized. Yet no efforts to contain the aerosols created by the machine were undertaken.[404] Reasonably available and widely used technology existed in March 1977 through which such containment could have been achieved without unduly frustrating the purposes of the Uni-Glatt experiments.[405]

The Uni-Glatt machine could have been placed inside a "glove box," which is a physical barrier that isolates the operator of a machine from the machine itself but allows the operator to insert his hands into gloves mounted on the inside and perform whatever tasks are associated with the operation of the machine.[406] Alternatively, the Uni-Glatt could have been placed within a "laminar flow containment hood," a device with a very high-efficiency filter capable of capturing microscopic viral particles before they escaped into the atmosphere of a laboratory.[407] Laminar flow hoods effectively limit a laboratory worker's exposure to the hazardous material with which he is working to the worker's

404. *See* text, *supra* at 1459 and 1463–1464; Exh. G-125H at 94 (Huffaker Deposition, February 1, 1983); *see also* Tr. at 202–03 (Debbie).

405. Tr. at 1047 (Hattwick).

406. Exh. G-125D at 93–95 (Fox Deposition).

407. Tr. at 1402 (Shope); *see* Tr. at 2353–54 (Baer). Use of laminar flow containment hoods is discussed in Lab Safety Manual, *supra* note 309, at I–42, I–43.

hands, which ordinarily would be gloved when placed in the area where the work was being conducted.[408] A laminar flow hood is ordinarily operated under negative air pressure, with the air pressure outside the containment unit being greater than that inside, and air is exhausted from the hood through the high-efficiency filter. The hood renders the atmosphere surrounding the work area virtually free of airborne viral particles.[409]

[17] In March 1977 sound laboratory practice required the physical confinement of airborne suspensions of live rabies virus so that aerosol exposures to that virus would be minimized.[410] Dr. Debbie failed to perform the Uni-Glatt experiment within a glove box or a laminar flow hood, or to utilize one of the other options available which would have provided physical containment of the rabies virus that was aero-

408. Tr. at 1332 (Clark).

409. Tr. at 1047 (Hattwick); *see also* Tr. at 176 (Debbie).

410. Tr. at 1841 (Winkler); Tr. at 1044–47 (Hattwick); Tr. at 2020–21 (Richardson).

solized during the March 29, 1977 experiment.[411] The failure to provide such containment constituted negligence, and this negligence was an actual and proximate cause of the exposure which resulted in plaintiff Jerome Andrulonis' infection and disease.[412]

411. Other options available to Debbie included performing the function accomplished by the Uni-Glatt in a machine that was airtight, something that was technologically feasible in March 1977, or operating the Uni-Glatt within containment facilities located in the CDC's laboratories in Georgia, wherein workers wear pressurized suits that isolated them from the environment of the room in which a particular experiment is performed. Tr. at 1402 (Shope).

412. The same conclusion is reached if the guidelines established in "Classification of Etiologic Agents," *supra* note 399, are followed. That document, published by the United States Department of Health, Education, and Welfare in conjunction with its subunit, the CDC, appears to incorporate principles of risk assessment, and was discussed extensively at trial. The publication was intended to "provide[ ] a standard for evaluating the hazards associated with various etiologic agents and define[ ] minimum safety conditions for their management without restricting or hampering bona fide microbiological investigations." *Id.* at 1; *see also* Tr. at 1387-88 (Shope). Etiologic agents are placed in four classes of increasing hazard, with Class 1 agents being the least hazardous and Class 4 agents requiring the most stringent precautions. The segregation of agents in this manner is accompanied by the caveat that "each investiga-

tor must use scientific judgment in interpreting the classification." "Classification of Etiologic Agents," *supra* note 399, at 1. For each class of agents, recommendations are made regarding the level of containment necessary to safely conduct laboratory work with the agents within that class. *See id.* at 9–11. The publication stresses that the degree of hazard associated with a particular laboratory project depends not only on the etiologic agent but also "the nature and kind of study in which it is being used." *Id.* at 1. The nature of an experiment can alter the classification within which the agent used should be placed. *See, e.g.*, Tr. at 1875–77 (Winkler). It is specifically noted that "[a]erosol studies ... markedly increase the hazard...." "Classification of Etiologic Agents," *supra* note 399, at 1.

The "Classification of Etiologic Agents" lists most strains of rabies virus as Class 2 agents. *Id.* at 8. Class 2 agents are generally deemed agents of ordinary potential hazard, since they are typically transmitted to a previously uninfected host by accidental injection or ingestion. Tr. at 1965 (Richardson). Under ordinary conditions the rabies virus is not spread readily in the environment but rather infects through a "very specific route," namely the bite or parenteral route. Tr. at 1802 (Winkler). The suspension of rabies virus in an aerosol, however, changes the nature of the risk created by the experimental use of that virus, Tr. at 1876–77 (Winkler), and this is particularly true when the virus suspension is highly concentrated, as was the ERA-BHK/21 virus strain used in the Uni-Glatt machine on March 29, 1977, since the airborne transmission of the rabies virus is highly dose-related. The court is persuaded that a highly concentrated rabies virus strain whose characteristics have been altered in unknown ways by multiple passages in different cell systems should be considered a Class 3 etiologic

It appears that two assumptions had been made by Dr. Debbie and the technicians working with him when the Uni-Glatt experiments were conducted in 1976 and 1977. First, it was assumed that because virus strains derived from the ERA vaccine, including ERA-BHK/21 strains, were "attenuated" modified live virus strains, they were incapable of causing disease in man.[113] Second, it was assumed that if any

agent when used in experiments in which that virus is aerosolized. *See, e.g.*, Tr. at 1390 (Shope); *see also* Tr. at 1965, 2021-22 (Richardson).

The "Classification of Etiologic Agents" recommends that when Class 3 agents are used in laboratory procedures, "[n]egative air pressure [should be] maintained at the site of work in a preparation cubicle or under a hood[,] [a]ir [should be] recirculated only after it has been adequately decontaminated through high efficiency filters," and "[p]ersonnel at risk [should be] immunized against agents for which immune prophylaxis is available." "Classification of Etiologic Agents," *supra* note 399, at 10. Only this last precaution—immunization—was taken with regard to the March 29, 1977 Uni-Glatt experimental runs. This alone was not enough to satisfy the minimum requirements of laboratory safety given the unknown nature of the particular rabies virus strain used and given the creation of substantial aerosols containing that virus while the Uni-Glatt machine was in operation.

413. Tr. at 384, 458 (Debbie); *see also* Exh. G-125H at 185-87 (Huffaker Deposition, February 2, 1983); Exh. G-125H at 5 (Huffaker Deposition, February 16, 1983).

hazard was presented by the virus strains used, the presence of serum antibodies in the workers exposed to the virus would be sufficient to prevent the onset of disease in the event of infection by the virus.[414] Both assumptions were tragically ill-founded.

The first assumption ignored the fact that a virus strain can only be considered "attenuated" with respect to a specific species, and that a virus strain that is attenuated with respect to one species can be lethal to another species, even if the latter species is closely related to that for which the strain is attenuated. Indeed, a virus strain is often (if not always) made attenuated for one species by making it extremely pathogenic for another.[415] The assumption that ERA-derived strains could not cause disease in man was particularly imprudent with regard to the virus Dr. Baer provided for the March 29, 1977 experiment, since little was known about that virus strain.

The second assumption was equally insupportable. Dr. Debbie indicated in his testimony that in conducting the Uni-Glatt experiments, immunization by the Lilly vaccine was virtually the only safety precaution taken.[416] While vaccination is an im-

414. *See* Tr. at 452–57 (Debbie); *see also* Tr. at 384 (Debbie).

415. *See* text, *supra* at 1439–1441.

416. Tr. at 384, 444, 457 (Debbie).

portant part of safe laboratory practice when exposure to an etiologic agent is possible, reliance on vaccination alone in the absence of other measures designed to minimize the likelihood of an exposure was unreasonable.[417] This was particularly true when aerosol exposures were threatened. Immunological protection against disease in the event of an aerosol exposure to an etiologic agent is but a second line of defense, secondary to the prevention of the exposure in the first place.[418] This is generally true regardless of the etiologic agent involved; it is particularly true when there is a threat of exposure to an etiologic agent against which serum antibodies might be ineffective.

To review, the court finds that Jerome Andrulonis was exposed to an aerosol containing an unusually high concentration of rabies viral particles during the course of the Uni-Glatt experiment conducted March 29, 1977. This exposure would have been avoided if Dr. Debbie had conducted that experiment in a manner consistent with what was considered reasonably safe labo-

417. Tr. at 1043 (Hattwick); *see also* Tr. at 1887 (Winkler) (Immunization is part of but not all of safe laboratory procedure).

418. Tr. at 1043–44 (Hattwick); Tr. at 1841 (Winkler); *see also* Tr. at 1885–87 (Winkler).

ratory practice in March 1977. The court has found that Andrulonis contracted the disease of rabies as a result of the aerosol exposure he experienced during the March 29 Uni-Glatt experiment. Expert testimony established that to a reasonable degree of scientific certainty, Andrulonis would not have contracted rabies if the aerosols produced during the March 29 experiment had been contained.[419] Therefore, the court finds that the injuries Andrulonis suffered as a result of his disease are attributable in part to negligence properly charged to NYSDOH.

B. *The Culpable Conduct Attributable to the Government*

The United States is liable under the FTCA for the tortious actions of its employees "under circumstances where the United States, if a private person, would be liable to the claimant in accordance with the law of the place where the act or omission occurred." 28 U.S.C. § 1346(b); *see also id.* § 2674. The Government argues that the conduct of Dr. Baer and the representations of Drs. Baer and Winkler relevant to this lawsuit have no "private analog" under New York law because of the "unique" role of the CDC "as a vehicle for coordinating the United States public health eradication activities." The court rejects this argument.

419. Tr. at 1419-20 (Shope); Tr. at 1095 (Hattwick).

[18] There are certain governmental functions for which it has been held that no private analog exists, but those functions ordinarily involve "quasi-legislative or quasi-adjudicative action by an agency of the federal government ... of the type that private persons could not engage in and hence could not be liable for under local law." *Jayvee Brand v. United States*, 721 F.2d 385, 390 (D.C.Cir.1983) (quoted in *C.P. Chemical Co. v. United States*, 810 F.2d 34, 37-38 (2d Cir.1987)). However, the fact that a certain activity performed by the Government is not performed in the private sector, either because of prohibitive costs, unprofitability, or other reason, does not mean the activity has no "private analog." Courts consistently "'impose[ ] liability upon the United States for its performance of activities that are not usually performed by private persons.'" *Wells v. United States*, 851 F.2d 1471, 1474 n. 2 (D.C.Cir. 1988) (quoting *Canadian Transport Co. v. United States*, 663 F.2d 1081, 1090 (D.C. Cir.1980)), *cert. denied*, — U.S. —, 109 S.Ct. 836, 102 L.Ed.2d 969 (1989); *compare Indian Towing Co. v. United States*, 350 U.S. 61, 64, 76 S.Ct. 122, 124, 100 L.Ed. 48 (1955). This is true even if the activity in question could be deemed a part of a "core governmental function." *Berkovitz v. United States*, 486 U.S. 531, 108 S.Ct. 1954, 1960 & n. 5, 100 L.Ed.2d 531 (1988).

In the case at bar, none of the challenged activities by Government employees involved quasi-legislative or quasi-adjudica-

tive functions. It is not difficult to imagine that private laboratories would engage in joint research projects with other investigators, give opinions to one another concerning the rabies virus, and supply other laboratories with virus strains for research purposes.[420] Therefore, the court finds that plaintiffs' allegations of negligence have corresponding "private analogs." The court now turns to the various theories under New York's substantive tort law that plaintiffs assert as bases for rendering a private person liable if he committed the acts and omissions with which Drs. Baer and Winkler are charged.[421]

420. *Compare* Tr. at 1409–11 (Shope).

421. Among those theories advanced by plaintiffs is the contention that Dr. Baer was negligent in passaging and concentrating the ERA-BHK/21 virus strain used in the March 29 Uni-Glatt experiment to a high titer and failing to "test" that virus strain before supplying it to Debbie. The concentration of the virus strain aerosolized would not have been relevant if Andrulonis had not been exposed to the aerosolized virus. The evidence indicates that it would have been possible to conduct the Uni-Glatt experiments safely if proper precautions, including the use of a physical containment system, had been taken. *See* text, *supra* at 1478–1479. The evidence introduced at trial is insufficient to support a finding of liability on the basis of these allegations. Moreover, there was no expert testimony establishing that it was unsafe not to "test" a virus strain before it was used in an

1. The Relationship Between the CDC and NYSDOH

Much of the evidence presented at trial focused on the relationship of the CDC and NYSDOH in general, and Drs. Baer and Debbie in particular, in pursuing wildlife immunization research projects in the 1970s. It was Baer who first interested Debbie in research involving the oral vaccination of foxes.[422] Before their meeting at Griffin in 1970, Debbie's experience working with the rabies virus was limited, despite his position with NYSDOH, and the

experiment; rather, it was unsafe to expose a human being to an aerosol containing an "untested" strain.

Plaintiffs have urged the court to hold Andrulonis to a reduced burden of proof due to his permanent loss of memory and resultant inability to testify. This rule governs death cases tried in New York courts and arguably would apply to the case at bar if tried in state court, given Andrulonis' permanent memory loss concerning events that occurred before April 1977. The court need not resolve any of the difficult issues that would be raised by the application of this rule to the case at bar since, as will be seen, Andrulonis meets ordinary standards of proof in prevailing on two distinguishable theories of recovery.

422. *See* text, *supra* at 1453.

rabies laboratory at Griffin at that time was strictly a diagnostic unit.[423] After the first experiments conducted by Debbie in which a virus strain supplied by Baer was administered orally to red foxes at Griffin, the two scientists remained in contact, seemed to coordinate their subsequent research efforts involving attempts to develop a feasible wildlife vaccination program, and jointly authored a number of articles regarding their research findings.[424] It was Baer who alerted Debbie to the existence of an enteric coating technique that had been used to develop a vaccine for swine, which led to Debbie's discovery of the air suspension coating process.[425] Baer often arranged funding for Debbie's research projects from the CDC,[426] and was able to devise a way of providing federal

423. Tr. at 92, 97–98 (Debbie).

424. *See* text, *supra* at 1453–1455.

425. *See* text, *supra* at 1454–1455.

426. *See, e.g.*, text, *supra*, at 1454 (CDC provided partial funding for Debbie's work attempting to administer liquid virus incorporated in baits to foxes and his studies involving the oral administration of commercial ERA vaccine to species other than foxes).

money for the Uni-Glatt experiments.[427] Baer provided the ERA-BHK/21 virus strain that was used in the March 29, 1977 Uni-Glatt experiment.

427. In the late spring or early summer of 1976, after WARF's Harlen Hall had demonstrated the Uni-Glatt machine to the staff at Griffin, Debbie traveled to Atlanta to meet with Dr. Baer. Tr. at 197 (Debbie). While in Atlanta, Debbie sought funding from CDC officials, including Baer, for continued research involving the Wurster air suspension process. Debbie lacked funds with which to rent the Uni-Glatt machine, which cost about $55 a day, Tr. at 198 (Debbie), and Baer agreed to make inquiries on Debbie's behalf. Baer discovered that money could not be made available to NYSDOH for the purpose of renting the Uni-Glatt machine, but learned that the CDC could supply Debbie with the funds he needed for his research indirectly. Tr. at 2056 (Baer). Baer informed Debbie that the CDC would supply the money needed by entering into a professional services contract with Debbie. Debbie would be obligated to prepare sugar nonpareils coated with rabies virus and supply those nonpareils to the CDC in return for payments made directly to Debbie, with which the NYSDOH scientist was able to rent the Uni-Glatt. Through this arrangement, Debbie was able to obtain funds from the CDC to conduct experiments with the Uni-Glatt in July 1976, in the Fall of 1976, and in March 1977. Tr. at 199-202 (Debbie); Tr. at 2055-59 (Baer); Exhs. G-10, G-11, G-12, 36, 44.

The close working relationship between the laboratories in Albany and Georgia in furtherance of the wildlife vaccination research raise two related theories under which it could be argued that Government liability could attach if New York law were applied absent the constraints of the sovereign immunity doctrine. First, given the cooperative efforts of the CDC and NYSDOH in their attempt to develop a bait containing rabies vaccine that could be distributed in the wild, it is arguable that these two entities (or the scientists employed by them) were engaged in a joint enterprise. Second, it might be suggested that Dr. Debbie effectively was an "employee" of the United States within the meaning of the FTCA who continued research on behalf of Baer after the CDC terminated its research concerning wildlife vaccination in 1976.[428] Neither theory has been pressed by plaintiffs in their post-trial papers. Nonetheless, since a great deal of the evidence presented at trial implicates these theories, the court will briefly address them here.

Under New York law, "a joint enterprise is an endeavor in which two or more persons unite to achieve a common purpose

428. Tr. at 995–96 (Hattwick); Tr. at 2204–06 (Testimony of Dr. Walter R. Dowdle); *see also* Tr. at 2287–88 (Baer).

under such circumstances that each has express or implied authority to act for all with respect to the control of the means or agencies employed to execute the plan...." *Fairbairn v. State*, 107 A.D.2d 864, 864–65, 484 N.Y.S.2d 682, 683 (3d Dept.), *aff'd*, 66 N.Y.2d 620, 495 N.Y.S.2d 32, 485 N.E.2d 239 (1985); *see also Connell v. Hayden*, 83 A.D.2d 30, 52–55, 443 N.Y.S.2d 383, 399–400 (2d Dept.1981); Prosser & Keeton, § 72, at 516–22. If this were a case involving only private parties, a finding of a joint enterprise would lead to the imputation of the negligence of one member of the enterprise to the other. *Fairbairn*, 107 A.D.2d at 864–65, 484 N.Y.S.2d at 683. In the case at bar, this would mean that if a joint enterprise between the CDC and NYSDOH was found, the negligence of Dr. Debbie would be chargeable to the CDC.

[19] In enacting the FTCA, however, Congress waived the Government's sovereign immunity only for cases where an employee of the United States acting within the scope of his employment causes injury through a "negligent or wrongful act or omission," 28 U.S.C. § 1346(b), and the Supreme Court has held that "[r]egardless of state law characterization, the Federal Tort Claims Act itself precludes the imposition of liability if there has been no negligence or other form of 'misfeasance or nonfeasance' ... on the part of [a] Government [employee]." *Laird v. Nelms*, 406 U.S.

797, 799, 92 S.Ct. 1899, 1901, 32 L.Ed.2d 499 (1972). Consequently, the Government cannot be held vicariously liable for the acts of any individual not considered an "employee" of the United States within the meaning of the FTCA, *see* 28 U.S.C. § 2671, even if that individual is engaged in what would be termed under state law a "joint enterprise" with a Government employee or agency. *Compare Alexander v. United States*, 605 F.2d 828, 832–34 (5th Cir.1979).

[20, 21] The second question raised by the evidence regarding the cooperative efforts of Baer and Debbie to develop a vaccine that could be distributed in the wild is whether Debbie can be deemed an "employee of the Government" under § 2671, thus rendering the Government liable for Debbie's negligent acts and omissions committed during the course of such "employment." 28 U.S.C. § 1346(b). Included within the Act's definition of an "employee of the Government" are "officers or employees of any federal agency," *id.* § 2671, and the definition of "federal agency" includes "corporations primarily acting as instrumentalities or agencies of the United States, but does not include any contractor with the United States." *Id.* A party under contract with the United States--Debbie contracted to supply the CDC with nonpareils containing rabies virus that were produced in the Uni-Glatt experi-

ments [429]—can be an "agency" of the United States, but only if that party's "day-to-day operations are supervised by the Federal Government." *United States v. Orleans*, 425 U.S. 807, 815, 96 S.Ct. 1971, 1976, 48 L.Ed.2d 390 (1976) (footnote omitted); *Loque v. United States*, 412 U.S. 521, 528, 93 S.Ct. 2215, 2220, 37 L.Ed.2d 121 (1973); *Falls Riverway Realty v. City of Niagara Falls*, 754 F.2d 49, 57 (2d Cir. 1985). The provision of federal monies to that party is not determinative. *Orleans*, 425 U.S. at 815, 96 S.Ct. at 1976. In the case at bar, the CDC did not exercise the necessary degree of control over the daily operations of Griffin's rabies laboratory at any point while research concerning wildlife vaccination was being conducted there, and specifically the CDC did not devise or direct any of the Uni-Glatt experiments conducted in 1976 and 1977.[430] In sum, the United States cannot be held liable for the tortious conduct of Dr. Debbie in connection with the March 29, 1977 Uni-Glatt experiment.

429. *See supra* note 427.

430. *See* text, *supra* at 1453-1454 and 1456.

2. Negligent Misrepresentation

[22] Plaintiffs maintain that the United States is liable for the injuries Jerome Andrulonis suffered because WARF officials were induced into recommending the Uni-Glatt machine to Debbie for the enteric coating experiments by opinions regarding the safety of ERA-derived virus strains that were negligently rendered by Drs. Baer and Winkler of the CDC. Plaintiffs assert that the Uni-Glatt machine was inadequate to safely accomplish the purposes of the enteric coating experiments. WARF officials had sought assurances about "the safety of handling the vaccine" before recommending the Uni-Glatt or agreeing to demonstrate how the machine was operated. Debbie forwarded the opinion letters written by Baer and Winkler which Debbie had requested in response to the concerns expressed by WARF.[431] The information provided in those letters may have been sufficient to advise someone of the hazards associated with ordinary laboratory exposures to the ERA vaccine (the focus of Winkler's letter) and its derivatives (discussed in Baer's letter). They did not, however, address possible dangers associated

431. *See* text, *supra* at 1455–1456.

with aerosols containing virus strains derived from the commercial ERA vaccine.[432]

432. The Government argues that under the FTCA, Andrulonis cannot maintain any claim against it based on the communication of misinformation to WARF. In enacting the FTCA, Congress did not waive the Government's sovereign immunity for claims sounding in misrepresentation and deceit. 28 U.S.C. § 2680(h). The Supreme Court has interpreted the "misrepresentation" exception to cover negligent as well as intentional misrepresentations. *United States v. Neustadt*, 366 U.S. 696, 703–06, 81 S.Ct. 1294, 1298–1300, 6 L.Ed.2d 614 (1961). In *Block v. Neal*, 460 U.S. 289, 103 S.Ct. 1089, 75 L.Ed.2d 67 (1983), the Court stated that "the essence of an action for misrepresentation, whether negligent or intentional, is the communication of misinformation on which the recipient relies." *Id.* at 296, 103 S.Ct. at 1093.

While this language suggests a broad reading of the misrepresentation exception of the FTCA, the courts have constricted it to claims that "fall[ ] within the commonly understood definition of a misrepresentation claim . . ." *Id.* at 296 n. 5, 103 S.Ct. at 1093 n. 5. As the Supreme Court noted in *Neustadt*, "many familiar forms of negligent conduct may be said to involve an element of 'misrepresentations,' in the generic sense of the word." 366 U.S. at 711 n. 26, 81 S.Ct. at 1302 n. 26 (citation omitted). For purposes of the exception provided in § 2680(h), however, "misrepresentation" has been "confined very largely to the invasion of interests of a financial or commercial character, in the course of business dealings.'" *Id.* (quotation omitted); *see also Block v. Neal*, 460 U.S. at 296 n. 5, 103 S.Ct. at 1093 n. 5. More recently, the Supreme Court has indicated that the misrepre-

sentation exception "relieves the Government of tort liability for *pecuniary* injuries which are wholly attributable to reliance on the Government's negligent misstatements." *Block v. Neal*, 460 U.S. at 297, 103 S.Ct. at 1094 (emphasis added). Most courts have interpreted the exception to apply only to "claims arising from *commercial* decisions based on false or inadequate information provided by the government." *Frigard v. United States*, 862 F.2d 201, 202 (9th Cir.1988) (emphasis added), *cert. denied*, — U.S. —, 109 S.Ct. 2448, 104 L.Ed.2d 1003 (1989); *Keir v. United States*, 853 F.2d 398, 411 (6th Cir.1988); *Kohn v. United States*, 680 F.2d 922, 926 (2d Cir.1982); *but see Wells v. United States*, 655 F.Supp. 715, 723–24 (D.D.C.1987), *aff'd on other grounds*, 851 F.2d 1471 (D.C.Cir. 1988), *cert. denied*, — U.S. —, 109 S.Ct. 836, 102 L.Ed.2d 969 (1989).

The injuries suffered in the present case were not "of a financial or commercial character" resulting from poor "commercial decisions based on false or inadequate information provided by the government." The focus of Andrulonis' claim is not "economic loss as a result of a commercial decision which was based on [either] false statements by the government or a failure to provide information which [the Government] had a duty to provide." *Green v. United States*, 629 F.2d 581, 584–85 (9th Cir. 1980). The opinion letters were written by Baer and Winkler in an effort to assure WARF officials of the safety of the virus strains to be used in the Uni-Glatt machine. In partial reliance on these letters WARF recommended the Uni-Glatt to Debbie. Therefore, the court finds that the "misrepresentation" exception is inapplicable.

Under New York law, "[l]iability in negligence may ... rest on some form of written misrepresentation or nondisclosure on the part of defendant by which plaintiff or a third party is misled, resulting in injury or damage to plaintiff." *Eiseman v. State*, 70 N.Y.2d 175, 187, 518 N.Y.S.2d 608, 614, 511 N.E.2d 1128, 1135 (1987). A plaintiff can recover for personal injuries as well as economic damages if such injuries were causally related to the negligent misrepresentation. *See id.* 518 N.Y.S.2d at 609–11, 614, 511 N.E.2d at 1130–32, 1135.

> Liability in such cases arises only where there is a duty, if one speaks at all, to give the correct information. And that involves many considerations. There must be knowledge, or its equivalent, that the information is desired for a serious purpose, that he to whom it is given intends to rely and act upon it; that, if false or erroneous, he will because of it be injured in person or property. Finally, the relationship of the parties, arising out of contract or otherwise, must be such that in morals and good conscience the one has the right to rely upon the other for information, and the other giving the information owes a duty to give it with care.

International Products Co. v. Erie R. Co., 244 N.Y. 331, 337–38, 155 N.E. 662, 664 (1927); *see also Eiseman*, 70 N.Y.2d at 187–89, 518 N.Y.S.2d at 613–14, 511 N.E.2d at 1134–35; *White v. Guarente*, 43 N.Y.2d 356, 362–63, 401 N.Y.S.2d 474, 478, 372 N.E.2d 315, 319 (1977).

[23] The court finds that the misleading letters drafted by Baer and Winkler do not form a basis for Government liability for the injuries sustained by Jerome Andrulonis.[433] A major premise for plaintiffs' argument is that the Uni-Glatt machine was inherently unsafe for use in coating rabies virus onto nonpareils because it was not airtight and operated under positive pressure, thus expelling aerosolized virus into the atmosphere surrounding the machine. The expert testimony concerning safe alternatives available for performing this experiment, however, did not foreclose altogeth-

433. The court will assume for the purposes of disposing of this claim that by negligently misleading WARF, Baer and Winkler breached a duty owed plaintiff Jerome Andrulonis. Whether the Government's duty to "give the correct information" would extend so far appears to be a close question under New York law. *See Ossining Union Free School District v. Anderson LaRocca Anderson*, 73 N.Y.2d 417, 541 N.Y.S.2d 335, 539 N.E.2d 91 (1989); *Eiseman*, 70 N.Y.2d at 187–89, 518 N.Y.S.2d at 614–15, 511 N.E.2d at 1135; *White*, 43 N.Y.2d at 362–63, 401 N.Y.S.2d at 478, 372 N.E.2d at 319.

er the use of the Uni-Glatt.[434] The evidence indicates that if the Uni-Glatt had been placed within either a glove box or laminar flow containment hood, a physical barrier between Andrulonis and the aerosolized rabies virus would have been erected and the experiments could have been safely conducted. In view of this, it cannot be said that WARF's provision of the machine was negligent; consequently, there is no basis for concluding that WARF would not have supplied the Uni-Glatt for the March 29, 1977 experiment if not for the misleading letters prepared by Baer and

434. In his deposition testimony, Dr. C. David Fox opined that the Uni-Glatt machine was defectively designed for use with the ERA-BHK/21 virus strain prepared by Baer for the March 29, 1977 coating experiment and that the recommendation of the machine for an experiment in which such a virus strain would be aerosolized constituted negligence. Exh. G-125D at 49 (Fox Deposition). Elsewhere, however, Dr. Fox indicated that it was possible to place a barrier between the machine and its operator. Exh. G-125D at 93-94 (Fox Deposition). There is other testimony also indicating that physical barriers could have been erected that would have severely limited if not eliminated entirely Andrulonis' exposure to aerosolized virus. Tr. at 1402 (Shope); *see also* Tr. at 1047 (Hattwick); Tr. at 1841 (Winkler); Mallison Report, *supra* note 221, at 3. On the basis of the record developed in this case, the court cannot conclude that the machine was inevitably unsafe for use in the experiments contemplated by Debbie given the availability of physical containment systems.

Winkler. Therefore, plaintiffs have failed to demonstrate that the negligent misrepresentations made by CDC personnel to WARF officials proximately caused the injuries suffered by Jerome Andrulonis.

3. Failure to Warn

At the request of Dr. Debbie, Dr. Baer prepared and delivered to Debbie an ERA-BHK/21 virus strain with a titer of 8.1 for use in a coating run with the Uni-Glatt machine. The strain Baer prepared was vastly more concentrated than any virus strain previously used in a Uni-Glatt experiment. At no time prior or subsequent to delivering the virus to Debbie did Baer warn any NYSDOH researcher or other employee of any potential danger associated with the use of that virus or of any hazard connected with an exposure to aerosols containing that virus strain, although the workers at Griffin's rabies laboratory were told that the virus was an ERA-BHK/21 strain with a titer of over 8.[435]

[24–27] Under traditional common law tort principles, there is no general duty to warn others of a foreseeable risk of harm

435. Exh. G-125C at 162-63 (Trimarchi Deposition).

even if one has knowledge of the risk that exists. Restatement (Second) of Torts § 314. This rule has been eroded, however, by a gradual extension of liability for "nonfeasance" to a group of relations for which custom or social policy requires the imposition of a duty of affirmative action. *Schumacher v. Richards Shear Co.*, 59 N.Y.2d 239, 247, 464 N.Y.S.2d 437, 441, 451 N.E.2d 195, 199 (1983). In certain cases, the existence of a "special relationship" will impose upon one party a duty "to warn another of known dangers, or, in some cases, of those dangers which he had reason to know." *Id.* at 246, 464 N.Y.S.2d at 441, 451 N.E.2d at 199.[436] The special rela-

436. The Government argues that plaintiff cannot assert a claim predicated on allegations concerning Baer's failure to warn Debbie of the hazardous nature of the ERA-BHK/21 virus strain used in the March 29, 1977 experiment by virtue of the "misrepresentation" exception of the FTCA. 28 U.S.C. § 2680(h). The court rejects this contention. As discussed above, the misrepresentation exception generally applies only in cases where the plaintiff has suffered a pecuniary loss as a result of a commercial decision made in reliance on misinformation or lack of information received from the Government. *See supra* note 432. Actions in which a plaintiff seeks to recover damages for personal injuries resulting from a defendant's failure to warn, in contrast, do not involve "business dealings of a financial or commercial character." *See Ingham v. Eastern Air Lines, Inc.*, 373 F.2d 227, 239 (2d Cir.), *cert. denied*, 389 U.S. 931, 88 S.Ct. 295, 19 L.Ed.2d 292 (1967).

Moreover, the failure to warn theory pressed against the Government in the case at bar is based on negligence theory: plaintiff alleges that Baer breached his duty of care by supplying Debbie with a product that was unreasonably dangerous. While it is maintained that the inherently dangerous product became *unreasonably* dangerous because Baer failed to warn Debbie of the hazards associated with it, the failure to communicate adequate information about the virus strain is not "essential" to plaintiff's claim, *see Block v. Neal*, 460 U.S. at 297, 103 S.Ct. at 1094, since liability on this theory could have been avoided altogether if Baer had refused to supply the agent or had not prepared a virus strain potentially pathogenic to man for use in the Uni-Glatt experiment. In other words, this is a case where a pure negligence claim has "overlap" on certain factual and legal questions with a common law claim for negligence misrepresentation. Under *Block v. Neal*, the former claim is not barred by the misrepresentation exception. *Id.* at 298, 103 S.Ct. at 1094. Other courts are in agreement that negligence claims predicated on a failure to warn theory are not precluded by § 2680(h). *See, e.g., Ramirez v. United States*, 567 F.2d 854, 856–57 (9th Cir.1977) (en banc) (failure to warn of risks attendant to surgery does not constitute "misrepresentation" within meaning of § 2680(h)); *Ingham v. Eastern Air Lines*, 373 F.2d at 238–39 (action against Government for failure of air traffic controllers to warn pilot of lack of visibility at airport not precluded by misrepresentation exception); *United Air Lines, Inc. v. Wiener*, 335 F.2d 379, 398 (9th Cir.) ("gravamen of the action is not misrepresentation but the negligent performance of operational tasks, although such negligence consisted partly of a failure of a duty to warn"), *cert. dismissed*, 379 U.S. 951, 85 S.Ct. 452, 13 L.Ed.2d 549 (1964).

tionship between the "supplier" of a "dangerous product" and those individuals who the supplier could reasonably foresee would use the product or otherwise be "exposed to a foreseeable and unreasonable risk of harm by the failure to warn" of hazards associated with foreseeable uses of the product is one which will give rise to a duty to warn. *McLaughlin v. Mine Safety Appliances Co.*, 11 N.Y.2d 62, 68, 226 N.Y.S.2d 407, 411, 181 N.E.2d 430, 433 (1962). Moreover, some courts have recognized that a "special relationship" exists between one whose conduct, even if non-negligent, has contributed to the creation of a hazard, and those individuals foreseeably imperiled by that hazard. *See* Prosser & Keeton § 56 at 377. The court will address in turn whether a duty to warn on the part of Dr. Baer arose out of either relationship and if so, whether that duty was breached, contributing to the injuries suffered by Andrulonis.

a. A Supplier's Duty to Warn

[28] The ERA-BHK/21 virus strain Baer provided to Debbie was known to be pathogenic to several animal species, and absent contrary evidence should have been assumed to be pathogenic to man.[137] Given

437. *See* text, *supra* at 1462 and 1475.

that its potential to cause human death was unknown, the virus strain Baer provided would be deemed a "dangerous product," and Baer had the same duty to warn that New York law imposes on a "supplier" of a dangerous product.

[29, 30] Initially, the court notes that Baer's duty to warn is in no way diminished or altered by the fact that he did not receive payment for preparing and delivering the ERA-BHK/21 virus strain to Dr. Debbie for the March 29 experiment. A donor of a chattel who either knows or should know that the chattel is dangerous for its intended or a foreseeable use is held to the same standard of care as a commercial supplier of such a chattel. *See* Restatement (Second) of Torts § 405; *Pease v. Sinclair Refining Co.*, 104 F.2d 183, 186 (2d Cir.1939) (applying New York Law); *Beede Waste Oil Corp. v. Recycling Industries, Inc.*, 533 F.Supp. 484, 486 (D.Mass. 1982) (applying Massachusetts law).[438]

438. In any event, the court finds that Baer's provision of the virus strain for use in the March 29 experiment was not wholly gratuitous. Baer derived benefits from Debbie's enteric coating research both because it furthered the wildlife immunization research to which he had committed himself, and because it provided him with enterically coated nonpareils containing rabies virus produced by the Uni-Glatt experiment of March 29. If some benefit, however amorphous, is expected to accrue to the party providing the chattel, that party has been deemed a "supplier" and held to the standard of care imposed on suppliers. *See Pease*, 104 F.2d at 186.

[31–35] A supplier's duty to warn arises out of New York's recognition that "[a] product can be dangerous even if it is not defectively designed." *Smith v. Hub Mfg., Inc.*, 634 F.Supp. 1505, 1508 (N.D.N.Y.1986). A supplier of a product who is aware or reasonably should be aware that the product is dangerous when put to a foreseeable use is charged with a duty "to exercise reasonable care to inform the user of the facts which make the product dangerous." *Young v. Elmira Transit Mix, Inc.*, 52 A.D.2d 202, 204–05, 383 N.Y.S.2d 729, 731 (4th Dept.1976) (Cardamone, J.) (citing Restatement (Second) of Torts § 388); *see also Billiar v. Minnesota Mining and Mfg. Co.*, 623 F.2d 240, 243 (2d Cir.1980) (applying New York law); *McLaughlin*, 11 N.Y.2d at 68, 226 N.Y.S.2d at 411, 181 N.E.2d at 433. If the supplier of the product has no reason to believe that one who foreseeably uses the product realizes the known dangers associated with its use and yet fails to adequately warn the user of those dangers, and if the failure to warn is a proximate cause of the user's injury, the user may recover against the supplier under basic negligence theory.[439]

439. Besides giving rise to a negligence cause of action, the New York courts have held that "inadequate warnings will render a product defective for purposes of warranty and strict products liability." *Ezagui v. Dow Chemical Corp.*, 598 F.2d 727, 733 (2d Cir.1979) (applying New

York law); *see also Robinson v. Reed-Prentice Division*, 49 N.Y.2d 471, 478–79, 426 N.Y.S.2d 717, 720, 403 N.E.2d 440, 443 (1980); *Torrogrossa v. Towmotor Co.*, 44 N.Y.2d 709, 711, 405 N.Y.S.2d 448, 449, 376 N.E.2d 920, 921 (1978). The primary distinction between a failure to warn case brought in negligence and one brought in strict tort liability for a product defect is that under the latter theory the plaintiff need not prove that the defendant "knew or should have known of the harmful character of the product without a warning." *Lancaster Silo & Block Co. v. Northern Propane Gas Co.*, 75 A.D.2d 55, 64, 427 N.Y.S.2d 1009, 1015 (4th Dept.1980). This effectively shifts the factfinder's focus from the fault of the defendant to the defect in the product allegedly caused by the inadequate warning. *See Caprara v. Chrysler Corp.*, 52 N.Y.2d 114, 123, 436 N.Y.S.2d 251, 255, 417 N.E.2d 545, 549 (1981). By enacting the FTCA, the United States waived its sovereign immunity only for cases where an employee of the Government acting within the scope of his employment causes injury through a "negligent or wrongful act or omission," 28 U.S.C. § 1346(b), and the Supreme Court has held that "[r]egardless of state law characterization, the Federal Tort Claims Act itself precludes the imposition of liability if there has been no negligence or other form of 'misfeasance or nonfeasance' ... on the part of the Government." *Laird v. Nelms*, 406 U.S. 797, 799, 92 S.Ct. 1899, 1901, 32 L.Ed.2d 499 (1972) (quoting *Dalehite v. United States*, 346 U.S. 15, 45, 73 S.Ct. 956, 972, 97 L.Ed. 1427 (1953)). Although *Laird* involved an attempt to impose absolute liability on the Government for the ultrahazardous activities of its employees, other courts have relied on it to foreclose recovery against the United States un-

See Kerr v. Koemm, 557 F.Supp. 283, 286 (S.D.N.Y.1983) (applying New York law); *Young*, 52 A.D.2d at 204–05, 383 N.Y.S.2d at 731 (citing Restatement (Second) of Torts § 388). If it is found that a duty to warn exists, both the nature of the warning that should be given and the identity of the parties to whom it should be given will depend in large part on the factual context within which the duty arises. Factors to be weighed include "the harm that may result from use of the product without the warnings, the reliability and adverse interest of the person to whom notice is given, the kind of product involved and the burden in disseminating the warning." *Frederick v. Niagara Machine & Tool Works*, 107 A.D.2d 1063, 1064, 486 N.Y.S.2d 564, 565 (4th Dept.1985); *see also Cover v. Cohen*, 61 N.Y.2d 261, 276, 473 N.Y.S.2d 378, 386, 461 N.E.2d 864, 872 (1984).

der a strict liability theory absent a showing of fault on the part of the Government's employee. *See, e.g., Gober v. United States*, 778 F.2d 1552, 1556–57 & n. 9 (11th Cir.1986); *Maltais v. United States*, 546 F.Supp. 96, 100 (N.D.N.Y.1982) (Miner, J.), *aff'd*, 729 F.2d 1442 (2d Cir.1983). Consequently, plaintiff Jerome Andrulonis cannot recover against the United States on the basis of Dr. Baer's failure to warn under a defective product theory, but instead must demonstrate that Baer would have been liable under negligence theory if he is to prevail on this aspect of his case.

In March 1977, Dr. George Baer arguably was the most knowledgeable rabies expert in the United States, and his status as a member of the community of rabies experts was unquestioned.[440] He was the editor of the only major modern treatise on the rabies virus that was in existence at that time.[441] Virtually every scientific publication that is cited in the background section of this opinion was either a chapter in Baer's treatise or cited in the treatise. Baer testified that in March 1977 he was familiar with the literature concerning the ERA-BHK/21 virus strain that has been discussed above.[442] Baer knew that the rabies virus can be transmitted through exposure to aerosols containing the virus,[443] and that airborne rabies virus probably infected animals, including humans, through the olfactory nerves in the nasal

440. Tr. at 1251 (Clark); Tr. at 1397-98 (Shope); Tr. at 2331 (Baer).

441. *See* Tr. at 1284 (Clark). That treatise was *Natural History*, *supra* note 9.

442. Tr. at 2302-08 (Baer); *see* text, *supra* at 1460-1463.

443. Tr. at 2309-13 (Baer).

epithelium.[444] While Baer indicated that he believed in March 1977 that antibodies induced by the Lilly vaccine would offer some protection against disease as a result of an aerosol exposure, he conceded that such protection had "not been shown definitely."[445] Specifically, Baer testified that in March 1977 "it was not known" whether an aerosol exposure to the ERA-BHK/21 virus strain was capable of causing the disease of rabies in a person with a demonstrable level of rabies antibodies at the time of exposure.[446] In short, the same level of knowledge about the rabies virus and its pathogenesis after an aerosol exposure is imputable to Dr. Baer as was imputed to Dr. Debbie by virtue of Baer's status as a rabies expert;[447] the evidence at trial convincingly demonstrated that Baer's *actual* knowledge was far superior to that of anyone employed in the rabies laboratory at Griffin.

444. Tr. at 2151, 2321 (Baer); *see also* "Airborne Rabies," *supra* note 97, at 119–20 (published in Baer's treatise). Baer also testified that he was familiar with the research establishing the primary importance of the olfactory region in the pathogenesis of airborne rabies. Tr. at 2318 (Baer).

445. Tr. at 2151–52 (Baer).

446. Tr. at 2255–56 (Baer).

447. *See* text, *supra* at 1474–1476.

[36, 37] Given Baer's knowledge of the virus strain he had prepared and the potential hazards associated with it, Baer had a duty to exercise reasonable care to insure that those who used or were exposed to that virus were aware of the facts that made it hazardous.[448] When he delivered the ERA-BHK/21 virus strain to Dr. Debbie on the evening of March 28, 1977, Baer informed Debbie that the virus strain he had prepared was an ERA-BHK/21 strain and that it had a titer of over 8.[449] The court finds that at the time this warning was given, it was adequate to discharge Baer's duty to warn.

[38] Initially, the court notes that although Baer's duty to warn extended to foreseeable users of the virus strain such as Andrulonis, established custom and practice among laboratories supplying etiologic agents did not require that the warning actually be given to Andrulonis himself. The proof at trial indicated that because it is not always possible to anticipate which laboratory worker at a particular laboratory would come into contact with an etiologic agent, the supplier of such an agent was expected to communicate infor-

448. *See* Tr. at 1304-05 (Shope).

449. *See* text, *supra* at 1463.

mation about its hazard to the scientist in charge of the experiment for which the agent was requested, with the understanding that the scientist in charge would assure that his workers were adequately informed.[450] The court believes that this accepted practice was reasonable, and thus it was reasonable for Baer to provide the relevant information about the virus strain to Debbie rather than directly to Andrulonis. *Compare Lindsay v. Ortho Pharmaceutical Corp.*, 637 F.2d 87, 91 (2d Cir. 1980) (New York law requires a manufacturer of a prescription drug to warn doctor, not patient, of potential dangers). In any event, it appears that Andrulonis was informed of the type of strain used in the March 29 Uni-Glatt experiment and the titer of that strain.[451]

With regard to the adequacy of the warning given March 28, 1977, a strong argument could be made that Baer reasonably assumed that the information he provided Debbie was sufficient to advise him of the hazards associated with exposing a laboratory worker to aerosols containing that virus.[452] It was arguably reasonable

450. Tr. at 1304–06 (Shope).

451. *See* text, *supra* at 1463.

452. *See* Tr. at 1445–47 (Shope).

to assume that Debbie, as the head of one of the country's better laboratories in the area of rabies research, was familiar with the basic principles, studies, and events relevant to the dangers of aerosolized rabies virus that have been discussed in this memorandum-decision. At least before witnessing the experiment conducted the next day, it was reasonable for Baer to assume on March 28 that Debbie was familiar with and adept at applying basic risk assessment principles in conceiving and conducting his experimental studies. When Baer told Debbie that he was providing an ERA-BHK/21 virus strain, the latter should have realized that it was not known whether or not this strain could cause catastrophic illness in man, and should have assumed it was potentially virulent. When Debbie was told of the virus strain's extremely high titer, he should have realized that the strain was highly concentrated, and thus posed a peculiar hazard in the event of an aerosol exposure. These facts alone should have alerted Debbie that the hazards associated with an experiment in which the virus strain supplied by Baer would be aerosolized required the employment of some physical containment system in order to meet the minimum standards of reasonably safe laboratory practice. On March 28, Baer had no reason to believe that such a containment system would not be used.

The fact that the initial warning given by Baer on March 28 may have been proportionate to the risk he reasonably perceived when he initially delivered the virus strain to Debbie, however, does not end the court's inquiry. Even if the warnings given at the time a product is delivered to its user adequately inform the user of "then known risks," risks subsequently revealed to the supplier may impose upon him a duty to warn of the subsequently discovered risk. *Cover v. Cohen*, 61 N.Y.2d at 275, 473 N.Y.S.2d at 385, 461 N.E.2d at 871. Before Andrulonis was exposed to aerosolized virus on March 29, 1977, Baer became aware or should have become aware of a risk involved in the manner the product he supplied was being used that would not have been apparent had he not been present during the March 29 experiment. Before a solution containing the ERA-BHK/21 strain Baer prepared had been introduced into the Uni-Glatt machine, Baer became aware of the fact that the Uni-Glatt machine, which he knew was not airtight, was not being operated within a laminar flow containment hood or other physical containment system. Baer testified that he knew before the virus was introduced into the Uni-Glatt that the machine would create an aerosol containing that virus.[453] The discovery of these facts

453. Tr. at 2350-51 (Baer); *see also* Tr. at 2218-19 (Dowdle).

should have alerted him to the risk that those working near the Uni-Glatt machine would be exposed to a heavy dose of potentially deadly live rabies viral particles, and that those workers were not assured of protection against the rabies disease by any vaccination regimen available in March 1977. This discovery gave rise to a duty to further warn Debbie of the hazards involved in the way he was using the ERA-BHK/21 virus.[454]

The court finds that in March 1977 Dr. Baer knew or should have known of the basic laboratory safety and risk assessment principles discussed above.[455] As head of the CDC's main rabies laboratory in Lawrenceville, Georgia, Dr. Baer was chargeable with knowledge of those principles to the same extent as Dr. Debbie. Moreover, the evidence established that all studies conducted in the CDC's laboratories in Georgia in which aerosols containing etiologic agents were produced were performed in laminar flow containment hoods.[456] Baer's own testimony indicates that he was aware that safe laboratory practice required the containment of aero-

454. *Compare* Tr. at 1400, 1442 (Shope); Tr. at 2220–23 (Dowdle); *see also* Tr. at 2352 (Baer).

455. *See* text, *supra* at 1476–1479.

456. *See* Tr. at 1047 (Hattwick); Tr. at 2020 (Richardson); Tr. at 2353–54 (Baer); *see also* Tr. at 2197–99 (Dowdle).

sols, although he maintained that he believed the Uni-Glatt itself was a "closed system" that prevented aerosols from escaping into the general atmosphere of the rabies laboratory at Griffin.[457]

The court also finds that notwithstanding his testimony to the contrary,[458] Baer knew the Uni-Glatt machine was not airtight, and that he knew this before the ERA-BHK/21 virus strain was introduced into the machine on March 29. There are at least four bases for this finding. First, the court accepts Dr. Debbie's testimony that he told Dr. Baer the machine leaked reddish dust during the third phase of a coating run before Baer came to Albany.[459] Second, the court finds that it is more probable than not that Baer saw nonpareils escape from the transparent column in which they were being circulated before the high-titer ERA-BHK/21 strain was aerosolized in the column.[460] Third, even if the court were to accept Baer's testimony that he did not see nonpareils leak from the column but instead saw them escape from the exhaust duct, it would not be reasonable to believe that repairing an opening in

457. *See* Tr. at 2285 (Baer).

458. *See, e.g.*, Tr. at 2263 (Baer).

459. *See* Tr. at 504, 481-83 (Debbie).

460. *See* text, *supra* at 1463-1464.

the duct with electrician's tape would make the Uni-Glatt into an airtight, "closed" system.[161] Fourth, the machine as depicted in the photographs admitted into evidence at trial simply does not appear airtight.[162] Since Baer apparently saw the machine assembled by hand before any live virus strain was introduced into it,[163] the court is hard-pressed to accept his testimony that he believed the machine was impermeable.

On the whole, Dr. Baer's testimony at trial lacked credibility. His demeanor was generally poor, and many of his statements concerning rabies virus strains in general and the ERA-derived strains in particular were misleading or disingenuous. His testimony concerning the March 29 experiment was particularly implausible. For instance, Baer stated that he did not see any reddish dust escape from the Uni-Glatt ma-

461. *See* Tr. at 2284–85 (Baer); *see* text, *supra* at 1463–1464.

462. Exhs. 51, 52, 53A-O; *see* text, *supra* at 1456–1458 (describing machine).

463. *See* Tr. at 2118 (Baer) (testimony that Baer saw the NYSDOH workers "load the machine" before virus introduced, a procedure which required some disassembly); *see also* Tr. at 2279 (Baer) (testimony that Baer saw machine partly disassembled when virus solution was not being sprayed onto nonpareils).

chine during the third phase of the coating run conducted March 29, 1977.[464] This is unlikely given subsequent tests performed on the machine indicating that leakage from the machine was substantial.[465] Indeed, in late April or early May 1977, Baer told Dr. Fred Clark of the Wistar Institute in Philadelphia that the Uni-Glatt machine "leaked like Hell," with nonpareils "popping" out of the machine and into the laboratory environment.[466] Baer denies telling Clark that the machine "leaked like Hell;"[467] the court does not believe him. Equally unconvincing is Baer's explanation that he told Clark that the nonpareils were "popping all over the place but within the machine."[468] Although none of these examples of incredible testimony by Dr. Baer directly relate to the fact Andrulonis must establish to prevail in this action—namely, that Baer knew the machine leaked *before* Andrulonis was exposed to aerosolized virus on March 29—they do tend to undermine Baer's credibility on more relevant testimony.

464. Tr. at 2132–33 (Baer); *see also* Tr. at 2262–63 (Baer).

465. Mallison Report, *supra* note 221, at 2.

466. Tr. at 1259–60 (Clark).

467. Tr. at 2281–82 (Baer).

468. Tr. at 2135 (Baer).

[39] Thus, the court does not believe Baer when he denied that Debbie told him before March 29, 1977 that the Uni-Glatt leaked red dust during the final stage of a coating run. The court does not believe Baer's testimony that he saw nonpareils escape from the machine's exhaust duct rather than the column before his virus strain was pumped into the column on March 29, 1977. The court does not believe that Baer thought the Uni-Glatt was a "closed system," given its appearance and the manner in which NYSDOH employees assembled it. Baer knew the machine leaked and that it was not being operated in a proper containment system before Andrulonis experienced any aerosol exposure to the virus strain Baer had supplied. At the time he became aware of these facts, a duty to supplement his original warning to Debbie arose. Baer should have alerted Debbie to the hazards the ERA-BHK/21 strain posed to laboratory workers by the manner in which Debbie intended to use it in carrying out his experiment. Baer's failure to give any additional warning was a breach of his duty to Andrulonis.

To recover damages, plaintiff Jerome Andrulonis must also establish that Baer's negligent failure to warn either Debbie or Andrulonis of the hazards associated with the use that was being made of the ERA-BHK/21 virus strain during the March 29 experiment was a proximate cause of the injuries Andrulonis suffered. *Belling v. Haugh's Pools Ltd.*, 126 A.D.2d 958, 958–

59, 511 N.Y.S.2d 732, 733 (4th Dept.1987). It has been held that proximate causation cannot be found when the dangers of a product are obvious or well-known (the "obviousness" exception), or if the user is actually aware of the dangerous nature of the product supplied (the "knowledgeable user" exception).[469] *Id.* at 959, 511 N.Y.

469. At one time, if a hazard was "obvious" or well known, the courts found that no duty to warn existed at all. *See Jiminez v. Dries & Krump Mfg. Co., Inc.*, 736 F.2d 51, 55 (2d Cir. 1984) (citing *Rosebrock v. General Electric Co.*, 236 N.Y. 227, 240–41, 140 N.E. 571, 575 (1923)). Then, in a passing reference, the New York Court of Appeals indicated that the fact that a defect in a product is open and notorious does not by itself negate the duty to warn. *Schumacher v. Richards Shear Co.*, 59 N.Y.2d 239. 249, 464 N.Y.S.2d 437, 442, 451 N.E.2d 195, 200 (1983); *see also Goussous v. Modern Food Market, Inc.*, 93 A.D.2d 417, 419–21, 463 N.Y.S.2d 550, 552 (3d Dept.1983); N.Y.P.J.I. 2:135 comment at 205 (Supp.1988) ("The obviousness of the danger or the discoverability of it upon inspection does not militate against a duty to warn, though it is a factor for the [factfinder] to take into account in assessing comparative fault."). The courts in *Schumacher* and *Goussous* reached this conclusion by analogizing to *Micallef v. Miehle Co.*, 39 N.Y.2d 376, 384 N.Y.S.2d 115, 348 N.E.2d 571 (1976), where the New York Court of Appeals held that in a design defect case, "a manufacturer is obligated to exercise that degree of care in his plan or design

so as to avoid any unreasonable risk of harm to anyone who is likely to be exposed to the danger when the product is used in the manner for which the product was intended ... as well as an unintended yet reasonably foreseeable use," *id.* at 385, 384 N.Y.S.2d at 121, 348 N.E.2d at 577–78, regardless of whether the defect was patent. *Id.* at 382–86, 384 N.Y.S.2d at 119–21, 348 N.E.2d at 576–77 (overruling *Campo v. Scofield*, 301 N.Y. 468, 95 N.E.2d 802 (1950)). The *Micallef* court held that the obviousness of a danger did not preclude a plaintiff from establishing a case for negligent design, but that such obviousness would be relevant to the comparative fault of that plaintiff. *Id.* at 387, 384 N.Y.S.2d at 122, 348 N.E.2d at 578.

The extension of *Micallef* to duty to warn cases creates some conceptual problems. Judge Leval noted in *Kerr v. Koemm* that "the rationale for eliminating the patent danger rule on design defects [does not] apply to the duty to warn. Obviousness should not relieve manufacturers of the duty to eliminate dangers from their design if that can reasonably be done, but obviousness relieves the manufacturer of a duty to inform users of a danger." 557 F.Supp. at 287 n. 1; *cf.* Prosser & Keeton, § 99, at 697–98 (knowability of risk should not be relevant to whether product is defectively designed, but is important in failure to warn case). In a dissent filed in *Schumacher*, Judge Jasen noted that "[t]he function of the warning is to make the product reasonably safe by alerting the user to the inherent danger of the product or the danger in misuse of the product." 59 N.Y.2d at 255, 464 N.Y.S.2d at 446, 451 N.E.2d at 204 (Jasen, J., dissenting). If the party in a position to avoid the injury was already aware of the knowledge the warnings should have conveyed, there is no reason to believe that the warning would have prevented the injury. The solution offered in *Belling*, cited in the text, was to restate the "obviousness" bar to recovery as an aspect of proximate cause rather than duty.

S.2d at 733. Further, an unforeseeable intervening act of negligence by a third party can defeat a finding of proximate cause. *Sheehan v. City of New York*, 40 N.Y.2d 496, 503, 387 N.Y.S.2d 92, 96, 354 N.E.2d 832, 835–36 (1976).

A determination of "the obviousness of the danger does not turn on the actual knowledge of the user," but instead depends on whether the supplier's "judgments about whether users will perceive the danger" on the basis of what was obvious or well known about the product absent warnings were objectively reasonable. *Kerr*, 557 F.Supp. at 287. The "obviousness exception" is inapplicable to the case at bar. Had Baer not actually witnessed the March 29 experiment, it might have been reasonable for him to assume that Debbie would appreciate the hazards associated with an aerosol exposure to the virus strain he supplied. Having witnessed the experiment, however, it should have become immediately apparent to Baer that Debbie in reality did not realize the hazards created by the manner in which he was using the high-titer ERA-BHK/21 virus strain.

[40, 41] The "knowledgeable user" exception is based on the observation that if the user knew of the danger associated with the use of a product in a particular way and proceeded to use the product in that way despite the known danger, the supplier's failure to warn could not have

been a proximate cause of the injury suffered by the user. *Kerr*, 557 F.Supp. at 286; *cf. Torrogrossa v. Towmotor Co.*, 44 N.Y.2d 709, 405 N.Y.S.2d 448, 449, 376 N.E.2d 920, 921 (1978) (where warning would not have prevented accident, failure to warn is not proximate cause of accident). Here, the court must inquire into the subjective knowledge of the injured party, determining "whether the particular user was aware of the danger." *Kerr*, 557 F.Supp. at 287 (citing *Rosebrock* and *McDaniel v. Williams*, 23 A.D.2d 729, 257 N.Y.S.2d 702 (1st Dept.1965)). Applying the "knowledgeable user" exception to the facts of this case, a distinction must be drawn between that knowledge that Debbie has had imputed to him and that knowledge he actually possessed in March 1977. As is discussed above,[470] Debbie assumed that because the ERA-BHK/21 virus strain was attenuated, it was incapable of causing disease in man. Further, Debbie assumed that the Lilly vaccine would in any event provide protection if an aerosol exposure to live virus capable of causing disease did occur. The court finds that Debbie did not possess actual knowledge of the danger posed by the virus strain Baer supplied. For reasons discussed in considerable detail below,[471] the court also finds that Andru-

470. *See* text, *supra* at 1480.

471. *See* text, *infra* at 1506–1507.

lonis did not ..ve actual knowledge of the danger created by the manner in which the ERA-BHK/21 virus strain was being used on March 29.

[42, 43] Finally, the negligence of Dr. Debbie is not a "superceding cause" of the injuries Andrulonis suffered, thereby breaking the chain of causation connecting Dr. Baer's negligent failure to warn and those injuries. Proximate cause is defined as that " 'which in a natural sequence, unbroken by any new cause, produces that event and without which that event would not have occurred.' " *Caraballo v. United States*, 830 F.2d at 22 (quoting *Rider v. Syracuse Rapid Transit Ry. Co.*, 171 N.Y. 139, 147, 63 N.E. 836 (1902)); *Ziecker v. Town of Orchard Park*, 147 A.D.2d 974, 975, 538 N.Y.S.2d 671, 672 (4th Dept.1989). The intervention of an act of a third party, whether negligent, reckless, intentional, or criminal, between a defendant's negligence and a plaintiff's injury does not constitute a "new cause" relieving the defendant of liability if the intervening act was "normal or foreseeable." *Woodling v. Garrett Corp.*, 813 F.2d 543, 555–56 (2d Cir.1987) (applying New York law and collecting cases). Rather, a "superseding cause" is an act by a third party that "interrupts the natural sequence of events, turns aside their course, prevents the natural and probable result of the original act or omission, and produces a different result that could not have been reasonably anticipated." *Sheehan*, 40 N.Y.2d at 503, 387 N.Y.S.2d at

96, 354 N.E.2d at 835 (quotation omitted). By attaching the flask containing the solution of the virus strain Baer supplied to the peristaltic pump of the Uni-Glatt and spraying the solution onto the nonpareils circulating in the machine's column without taking precautions designed to insure that workers were not exposed to the aerosols that escaped from the machine, Debbie and Andrulonis merely carried out the act that brought to fruition the hazard Baer should have anticipated and warned about.[472] Debbie's negligence was foreseeable, and

472. The Government argued that it cannot be held liable for the injuries suffered by Andrulonis as a supplier of a dangerous product because there was a "modification" of the ERA-3HK/21 virus strain that substantially altered it after it left "the possession and control" of Dr. Baer. *See Robinson v. Reed-Prentice Division*, 49 N.Y.2d 471, 479-81, 426 N.Y.S.2d 717, 720-21, 403 N.E.2d 440, 443-44 (1980). The court rejects this argument. First, there is no evidence that the virus strain left the control of Dr. Baer; indeed, Baer's own testimony is to the contrary. Tr. at 2357-58 (Baer). Second, there is no evidence that by aerosolizing the virus during the March 29 Uni-Glatt experiment in Baer's presence, Debbie was making an unintended use of the product Baer prepared and supplied.

does not relieve the Government of liability for Baer's negligence.

In sum, Baer's negligent failure to warn was a contributing cause to the aerosol exposure Andrulonis experienced. The court finds that if Baer had warned Debbie or Andrulonis of the danger created by the manner in which they were using the virus strain he supplied, more likely than not the aerosol exposure would not have occurred and Andrulonis would not have contracted the disease of rabies. The Government is answerable for Baer's negligence, and is liable for the injuries suffered by Jerome Andrulonis.

b. The Non-Negligent Creation of a Hazard

[44] Apart from his role as supplier of a dangerous product, Baer had a duty to warn Debbie or Andrulonis of the hazard to which Andrulonis was exposed during the March 29 Uni-Glatt experiment because his actions contributed to the creation of the hazard to which Andrulonis was exposed. Baer did not act negligently in preparing the extraordinarily concentrated virus strain used during the experiment or in providing the strain to Debbie for experimental use. By doing so, however, his affirmative actions contributed to the creation of a hazard, however innocently. When he witnessed the Uni-Glatt experiment on March 29, Baer should have immediately become aware of that hazard—that is, that Andrulonis was being exposed to

aerosols containing the highly concentrated and potentially pathogenic virus strain Baer had prepared.

[45] The Restatement (Second) of Torts provides that "[i]f the actor does an act, and subsequently realizes or should realize that it has created an unreasonable risk of causing physical harm to another, he is under a duty to exercise reasonable care to prevent the risk from taking effect." *Id.* § 321(a). This rule applies even if the original act is innocent. *Id.* § 321 comment a. The court is aware of only one case in which a New York court has applied this principle. *See Slavin v. State*, 249 A.D. 72, 291 N.Y.S. 721 (3d Dept.1936).[473] Nonetheless, the Restatement rule seems consistent with the policy considerations which help define the duties owed under New York tort law.

473. In that case, the State of New York was held liable for injuries suffered by the plaintiff during an arrest after a state trooper had negligently failed to relay a message to local police authorities cancelling a prior message indicating that the occupants of the plaintiff's automobile were suspected of armed robbery. The court did not indicate that the original message was negligently issued.

[46] It is well established that if an actor creates a dangerous condition through his own negligence, a relation has arisen which imposes on that actor a duty to give adequate warning of the presence of the dangerous condition and to correct it. Note, *Torts: Nonnegligent Participant Liable for Creation of Hazard*, 51 Minn.L.Rev. 362, 363 (1966) (hereinafter *Nonnegligent Participant*); Prosser & Keeton § 56, at 377. Liability in such cases was based on the original negligence causing the dangerous condition rather than the subsequent failure to give adequate warnings. *Nonnegligent Participant*, 51 Minn.L.Rev. at 363. When a dangerous condition is created by innocent conduct, "original negligence" upon which liability could be based is nonexistent, and early cases refused to find that a duty to warn independent of any affirmatively negligent conduct by the actor arose in such situations. *See* Prosser & Keeton § 56, at 377. The clear trend in more recent cases, however, is to impose upon the non-negligent actor who creates or contributes to the creation of a hazardous condition a duty to use reasonable care to warn others foreseeably endangered by the condition created. *See, e.g., Galanti v. United States*, 709 F.2d 706, 709 (11th Cir.1983) (applying Georgia law), *cert. denied*, 465 U.S. 1024, 104 S.Ct. 1279, 79 L.Ed.2d 683 (1984); *United States v. Aretz*, 248 Ga. 19, 26, 280 S.E.2d 345, 350 (1981); *Zylka v. Leikvoll*, 274 Minn. 435, 144 N.W.2d 358

(1966); *Chandler v. Forsyth Royal Crown Bottling Co.*, 257 N.C. 245, 125 S.E.2d 584 (1962); *Montgomery v. National Convoy & Trucking Co.*, 186 S.C. 167, 195 S.E. 247 (1938); *Simonsen v. Thorin*, 120 Neb. 684, 234 N.W. 628 (1931).

The delineation of the bounds of the public duty of an individual who exposes another to the risk of harm is dependent on social policy as much as "logic and science." *DeAngelis v. Lutheran Medical Center*, 58 N.Y.2d 1053, 1055, 462 N.Y.S.2d 626, 627, 449 N.E.2d 406, 407 (1983). Justifications for the traditional reluctance of courts to impose affirmative duties to warn or otherwise act to prevent injury to another include the impracticability of imposing such duties and a recognition of a defendant's interest in "keeping to himself" and in not being inconvenienced by a requirement to act for the prevention of harm to others. *See* Note, *Affirmative Duty After Tarasoff*, 11 Hofstra L.Rev. 1013, 1020–24 (1983). Neither of these policy justifications outweigh society's interest in preventing avoidable injuries and providing a remedy to those injured when otherwise innocent affirmative conduct by a defendant has contributed to the creation of a danger. In such cases, by limiting liability to those whose actions have created a hazard, an unmanageable extension of tort liability is avoided. Further, to the extent that the defendant had already decided to act in the manner he did in creating the danger, his interest in self-autonomy is diminished in

importance since he has "already injected himself into the plaintiff's realm." *Id.* at 1025; *compare Moch Co. v. Rensselaer Water Co.*, 247 N.Y. 160, 167, 159 N.E. 896, 898 (1928).

The court has found that Dr. Baer had actual knowledge of scientific principles about the rabies virus and about laboratory safety that should have alerted him immediately to the dangers associated with the unsafe manner in which the March 29 Uni-Glatt experiment was being performed. By preparing and then providing an extraordinarily concentrated and potentially pathogenic live rabies virus strain specifically for use in this experiment, Baer contributed to the creation of a hazardous condition in Griffin's rabies laboratory on March 29. Under these circumstances, Baer had a duty to warn Debbie and the other NYSDOH employees of the danger associated with the manner in which the virus product he created was being used. Baer's failure to warn was a concurring cause of Andrulonis' exposure to the rabies virus that day, and is an independent basis upon which the Government can be found liable in this case.

4. Baer's Duty to Intercede and Stop the Uni-Glatt Experiment

[47] "[C]ontrol of a dangerous instrumentality may call for care to stop its continuing [use] which is seen to threaten plaintiff." 3 F. Harper, F. James, & O. Gray, *The Law of Torts* § 18.6, at 722 (2d

ed. 1986). Independent of his duty to warn, Dr. Baer had a legal obligation to intercede and stop the March 29 experiment before Andrulonis was exposed to aerosols containing the ERA-BHK/21 virus strain. Baer testified that he could have withheld the ERA-BHK/21 strain at any point during the course of the March 29 experiment,[474] indicating that he retained control over the virus even after he gave it to Debbie the prior evening. For the reasons discussed above, the court finds that Baer should have been aware of the danger that was created when the virus was propelled through the Uni-Glatt machine during that experiment, and that he should have comprehended this danger before the virus was actually introduced into the machine. Under these circumstances, the court finds that Baer's failure to intercede and attempt to halt the experiment before Andrulonis was exposed to aerosols containing the virus was negligence for which Andrulonis can recover.

[48] This negligence theory is akin to the doctrine of "negligent entrustment." Under that theory, liability is imposed on "one who places in another's hands an instrumentality capable of doing serious harm if misused while knowing or having strong reason to believe that it will be

474. Tr. at 2357-58 (Baer).

misused to the detriment of others." *Robinson v. Reed-Prentice Division*, 49 N.Y.2d 471, 484, 426 N.Y.S.2d 717, 723, 403 N.E.2d 440, 446 (1980) (Fuchsberg, J., dissenting); *see also Splawnik v. Di Caprio*, 146 A.D.2d 333, 540 N.Y.S.2d 615 (3d Dept. 1989); Restatement (Second) of Torts § 390.[475] In these cases, "the duty of reasonable care is breached when one passively permits a danger to be created by supplying [a] product to a probably negligent user; the negligence or misuse by the user is considered to be but a foreseeable intervening cause of the injury." *Robinson v. Reed-Prentice Division*, 49 N.Y.2d at 484, 426 N.Y.S.2d at 723, 403 N.E.2d at 446 (Fuchsberg, J., dissenting); *see also Fredericks v. General Motors Corp.*, 48 Mich.

475. The text of the Restatement rule is as follows:

> One who supplies directly or through a third person a chattel for the use of another whom the supplier knows or has reason to know to be likely because of his youth, inexperience, or otherwise, to use it in a manner involving unreasonable risk of physical harm to himself and others whom the supplier should expect to share in or be endangered by its use, is subject to liability for physical harm resulting to them.

Restatement (Second) of Torts § 390. As is elaborated in the comments to that section, an actor "may not assume that human beings will conduct themselves properly if the facts which are known or should be known to him should make him realize that they are unlikely to do so." *Id.* § 390 comment b.

App. 580, 211 N.W.2d 44 (3d Div.1973) (applying Michigan law). In the case at bar, the "misuse" of the ERA-BHK/21 virus strain was the failure to properly contain aerosolized virus created during the Uni-Glatt experiment. As a witness to the March 29 experiment, Baer obviously had "strong reason to believe" that the NYS-DOH workers were misusing the virus product he created and supplied. Under these circumstances, Baer had a duty to at least attempt to stop the experiment before harm resulted. Because he retained control of the virus product, Baer's failure to intervene can fairly be deemed a direct and proximate cause of the damages Andrulonis suffered.

5. The Discretionary Function Exception

The Government has renewed its argument that the claims of Jerome Andrulonis are barred by the "discretionary function" exception to the FTCA. 28 U.S.C. § 2680(a). The court has previously addressed the applicability of this exception in the context of motions to dismiss and for summary judgment, rejecting the Government's contention that the conduct of Dr. Baer that gave rise to this lawsuit was protected by § 2680(a). *See Andrulonis v. United States*, 593 F.Supp. 1336 (N.D.N.Y. 1984). In rejecting the Government's position, the court viewed the record in the light most favorable to plaintiffs. The court now reassesses the Government's argument on the basis of the facts established at trial.

The discretionary function exception provides that the Government's waiver of sovereign immunity through that Act does not extend to "[a]ny claim ... based upon the exercise or performance or the failure to exercise or perform a discretionary function or duty on the part of a federal agency or an employee of the Government, whether or not the discretion involved be abused." 28 U.S.C. § 2680(a). The Supreme Court has observed that this exception "marks the boundary between Congress' willingness to impose tort liability upon the United States and its desire to protect certain governmental activities from exposure to suit by private individuals." *United States v. S.A. Empresa de Viacao Aerea Rio Grandense (Varig Airlines)*, 467 U.S. 797, 808, 104 S.Ct. 2755, 2762, 81 L.Ed.2d 660 (1984).

[49] While the Act does not expressly define what a "discretionary function or duty" is, it is clear that the mere fact that the Government employee whose conduct gave rise to the FTCA action "exercise[d] *some* judgment in the carrying out of his responsibilities cannot be determinative, or the discretionary function exception would swallow the general rule permitting tort suits against the government." *Andrulonis v. United States*, 593 F.Supp. at 1339 (emphasis in original) (citing, *inter alia*, *Caban v. United States*, 671 F.2d 1230, 1232 (2d Cir.1982)). Recent case law interpreting the exception "draw[s] the elusive line between protected and unprotected ac-

tivities" by focusing on whether the "action involved the balancing of policy factors." *Caban*, 671 F.2d at 1232. The Supreme Court has recently reiterated the inquiry that must be made:

> [A]ssuming the challenged conduct involves an element of judgment, a court must determine whether that judgment is of the kind that the discretionary function exception was designed to shield. The basis for the discretionary function exception was Congress' desire to "prevent judicial 'second-guessing' of legislative and administrative decisions grounded in social, economic, and political policy through the medium of an action in tort." *United States v. Varig Airlines*, [467 U.S. at 814, 104 S.Ct. at 2765]. The exception, properly construed, therefore protects only governmental actions and decisions based on considerations of public policy. See *Dalehite v. United States*, [346 U.S. 15, 36, 73 S.Ct. 956, 968, 97 L.Ed. 1427 (1953)] ("Where there is room for policy judgment and decision there is discretion"). In sum, the discretionary function exception insulates the Government from liability if the action challenged in the case involves the permissible exercise of policy judgment.

Berkovitz v. United States, 486 U.S. 531, 108 S.Ct. 1954, 1963, 100 L.Ed.2d 531 (1988). Put another way, the discretionary function exception shields the United States from liability for decisions to act in

accordance with a certain social, economic, or political policy, but "once [the Government] decides to act, it is responsible for its actions that are negligently carried out." *Caraballo v. United States*, 830 F.2d 19, 21 (2d Cir.1987).

The distinction drawn between discretionary acts involving policy choices that are immune from suit under the FTCA and those discretionary acts which can be the basis of a negligence action under the Act is more easily grasped by examining the holdings of leading cases. For example, in *Indian Towing Co. v. United States*, 350 U.S. 61, 76 S.Ct. 122, 100 L.Ed. 48 (1955), the Supreme Court found that the United States Coast Guard's decision whether to operate a lighthouse was discretionary, but that "once it exercised its discretion to operate a light ... and engendered reliance on the guidance afforded by the light, it was obliged to use due care to make certain that the light was kept in good working order," and that the failure to do so would render the United States liable under the FTCA. *Id.* at 69, 76 S.Ct. at 126–27. In *Caraballo*, the Second Circuit held that a decision whether to patrol a public beach was a "discretionary decision" for the United States Park Service to make, but once made the Park Service had a "non-discretionary duty" to carry those patrols out non-negligently. 830 F.2d at 22. In *Eklop Marine Corp. v. United States*, 762 F.2d 200 (2d Cir.1985), the Second Circuit ruled

that the Coast Guard's decision "to place a navigational aid at a particular location or to employ only one such aid at that location, as opposed to the initial decision to mark [an] obstruction [to navigation caused by a reef], is not an expression of any 'policy' of which [the court was] aware...." *Id.* at 205. Similarly, in *Drake Towing Co., Inc. v. Meisner Marine Const. Co.*, 765 F.2d 1060 (11th Cir.1985), the Eleventh Circuit also distinguished between the Coast Guard's decision to establish navigational aids in a particular area, which is discretionary, and the maintenance and operation of such aids in a non-negligent manner, which is not. *Id.* at 1064.

[50, 51] In the case at bar, doubts about the wisdom of the enteric coating project in general were raised during the course of the trial.[476] Governmental liability under the FTCA cannot be founded upon Dr. Baer's decision to encourage and aid NYS-

476. For instance, it was revealed that the CDC had discontinued wildlife vaccination research conducted in its Georgia laboratories because of safety concerns regarding the distribution of any live vaccine, if developed, in the wild. Tr. at 995–96 (Hattwick); *see also* Tr. at 2287–88 (Baer) (cross-examination by plaintiffs' counsel impliedly questions whether human health risk posed by rabies disease in the United States justified oral vaccination project); Tr. at 2205–06 (Dowdle) (same).

DOH in the furtherance of this research, for that decision implicated precisely the sort of public policy considerations contemplated by the discretionary function exception. Dr. Baer's failure to conduct a safety inspection of Griffin before supplying the high-titer virus strain used in the March 29, 1977 Uni-Glatt experiment was also questioned. This omission, however, was shown to be consistent with the general practice of the CDC to provide etiologic agents to research laboratories without routinely conducting safety inspections of those laboratories.[477] This practice is also immunized by the discretionary function exception. *Compare Varig Airlines*, 467 U.S. at 814–20, 104 S.Ct. at 2764–68 (Federal Aviation Administration's selection of a "spot-checking" method of enforcement of its safety regulations implicated policy considerations and thus could not form the basis of an FTCA action).

[52] The liability of the United States, however, is not grounded on the allegations that these policies were unsound. The court has found that Dr. Baer was negligent in failing to warn NYSDOH workers of the hazards associated with the ERA-BHK/21 virus strain that he prepared for the March 29 experiment, and that Baer was negligent in allowing the virus he had

477. Tr. at 1952–58 (Richardson); Tr. at 2051–53, 2067–69, 2263–64 (Baer).

prepared to be used in the Uni-Glatt experiment when he should have known that the conditions under which the experiment was being conducted were unsafe. Whatever choices made or discretion exercised by Baer in connection with the provision of the ERA-BHK/21 virus to NYSDOH without accompanying warnings or allowing that virus to be used in an experiment that Baer should have known was being conducted unsafely, those choices did not involve the weighing of public policy considerations. Instead, they involved "the kind of judgment that requires the knowledge and professional expertise of [the] government employees who implement government policies," *Aslakson v. United States*, 790 F.2d 688, 693 (8th Cir.1986), and thus fall outside the purview of the discretionary function exception.

[53] The Government argues that in prior rulings denying motions to dismiss under Rules 12(b)(6) and 56 of the Federal Rules of Civil Procedure, the court erroneously adopted a *per se* rule precluding the application of the discretionary function exception when the exercise of professional or scientific judgment on the part of employees of the United States was involved. *See Andrulonis v. United States*, 593 F.Supp. at 1339 (citing *Griffin v. United States*, 500 F.2d 1059, 1066 (3d Cir.1974)). The court did not intend to embrace any such *per se* rule by agreeing with the Third Circuit that conduct of Government em-

ployees involving "only performance of scientific evaluation and not the formulation of policy" is not "immunized from judicial review as a 'discretionary function.'" *Griffin*, 500 F.2d at 1066. The court did not and does not understand *Griffin* to imply that all professional and scientific decisions are beyond the reach of the discretionary function exception. *Accord Alabama Electric Co-Operative, Inc. v. United States*, 769 F.2d 1523, 1529 n. 2 (11th Cir.1985). Rather, "the fact that the decision is one involving professional discretion is less important than the question of whether or not the professional discretion involves policy considerations." *Id.*; *see also Wells v. United States*, 851 F.2d 1471, 1476–77 (D.C.Cir.1988), *cert. denied*, — U.S. —, 109 S.Ct. 836, 102 L.Ed.2d 969 (1989) (indicating that a decision based upon scientific considerations is protected by discretionary function exception *if* economic, social, and political policy considerations were also involved in decision). This reading is consistent with the Supreme Court's recent *Berkovitz* decision, and the court will continue to adhere to its prior interpretation of the exception.

The Government suggests that Dr. Baer exercised discretion implicating policy considerations by deferring to the judgment of the State scientists in charge at Griffin regarding the best means of handling the high-titer virus solution he supplied in the

Uni-Glatt experiment of March 29, 1977. The court does not accept the Government's contention that Baer's failure to attempt to halt an experiment that he should have known was being conducted in an unsafe manner was based on "political considerations favoring the preservation of a strong collegial relationship between CDC and state health departments." [478] There is no compelling evidence of the existence of such a "political policy." Indeed, one CDC employee who testified at trial indicated that if an observer of an experiment actually knows that the manner in which the experiment was being conducted endangered human life, intervention would have been appropriate.[479] This contention by the Government appears to be a *post hac* rationalization for the negligence of its employee.

478. Doc. 611 at 5.

479. Tr. at 2220–23 (Dowdle).

6. Summary

The court finds that the injuries Jerome Andrulonis has suffered as a result of contracting the disease of rabies were proximately caused by the negligence of an employee of the United States. Dr. Baer breached his duty to adequately warn either Debbie or Andrulonis of the hazards associated with the virus strain he supplied for use in the March 29, 1977 Uni-Glatt experiment. Further, Baer negligently failed to exercise proper control over a dangerous instrumentality by failing to intervene in an attempt to halt the March 29 Uni-Glatt experiment, and this failure was a contributing cause to the injuries Andrulonis ultimately suffered.

C. *The Culpable Conduct Attributable to Lilly and Thompson & Sons*

The Government seeks to diminish its liability proportionately by the equitable share of defendants Lilly and Thompson & Sons, who entered into settlement agreements with plaintiffs during the course of the trial. *See* N.Y.C.P.L.R. §§ 1401–1404 (McKinney 1976); N.Y.Gen.Oblig.Law § 15–108 (McKinney 1978). The Government claims that Lilly and Thompson & Sons are strictly liable for defects in the Lilly vaccine that they marketed. The researchers at NYSDOH relied upon DEV to provide protection against disease after infection by the rabies virus, and Jerome

Andrulonis developed the disease of rabies despite maintaining a reasonable level antibodies in his blood stream. The Government argues that the defective nature of DEV was a substantial cause of Andrulonis' illness.

Strict tort liability for the marketing of a product will be imposed if:

> (1) the product is "defective" because it is not reasonably safe as marketed; (2) the product was used for a normal purpose; (3) the defect was a substantial factor in causing the plaintiff's injuries; (4) the plaintiff by the exercise of reasonable care would not have both discovered the defect and apprehended its danger; [and] (5) the plaintiff would not have otherwise avoided the injury by the exercise of ordinary care.

Wolfgruber v. Upjohn Co., 72 A.D.2d 59, 61–62, 423 N.Y.S.2d 95, 97 (4th Dept.1979), *aff'd*, 52 N.Y.2d 768, 436 N.Y.S.2d 614, 417 N.E.2d 1002 (1980) (citing *Codling v. Paglia*, 32 N.Y.2d 330, 342, 345 N.Y.S.2d 461, 469, 298 N.E.2d 622, 628 (1973)). Strict liability can be charged to a manufacturer of a "defective" product "regardless of privity, foreseeability, or due care." *Sukljian v. Charles Ross & Son Co.*, 69 N.Y.2d 89, 94, 511 N.Y.S.2d 821, 823, 503 N.E.2d 1358, 1360 (1986). Retailers and distributors of defective products who regularly sell those products in the normal course of their business are also subject to strict tort liability. *Id.* at 95, 511 N.Y.S.2d at 823, 503 N.E.2d at 1360. Defendant Lilly was the

manufacturer of the lots of DEV from which Andrulonis received all of his rabies booster shots, and defendant Thompson & Sons was the distributor that sold the Lilly vaccine to NYSDOH. There is no legal distinction between these defendants under the facts of this case, and thus the court's discussion of the culpable conduct of Lilly applies with equal force to Thompson & Sons.

[54, 55] Turning to the first element of the test set out by Judge Cardamone in *Wolfgruber*, a product is deemed "defective" if it has a manufacturing flaw, a design defect, or is marketed without adequate warnings for the use of the product. *Sage v. Fairchild-Swearingen Corp.*, 70 N.Y.2d 579, 523 N.Y.S.2d 418, 421, 517 N.E.2d 1304, 1307 (1987); *Voss v. Black & Decker Mfg. Co.*, 59 N.Y.2d 102, 106–07, 463 N.Y.S.2d 398, 401, 450 N.E.2d 204, 207 (1983). There is no proof that any of the vaccine booster shots administered to Andrulonis were defectively made. Nor is the evidence adequate to establish that DEV was defectively designed. A defectively designed product is one that "is in a condition not reasonably contemplated by the ultimate consumer and is unreasonably dangerous for its intended use; that is, one whose utility does not outweigh the danger inherent in its introduction into the stream of commerce." *Robinson v. Reed-Prentice Division*, 49 N.Y.2d at 479, 426 N.Y.S.2d at 720, 403 N.E.2d at 443. Whether a product is defectively designed turns on "a balancing of the likelihood of harm against

the burden of taking a precaution against that harm," *Sage*, 70 N.Y.2d at 586, 523 N.Y.S.2d at 421, 517 N.E.2d at 1307, an inquiry that requires consideration of reasonably feasible alternative designs. *Voss*, 59 N.Y.2d at 109, 463 N.Y.S.2d at 402–03, 450 N.E.2d at 208–09. The weight of the evidence indicates that the Lilly vaccine was safe and effective in protecting humans from commonplace exposures to the rabies virus. There is no evidence that a feasible alternative to DEV existed in 1976 and 1977 that would protect someone against less common aerosol exposures to rabies virus; indeed, it may not even be possible to manufacture a rabies vaccine that would effectively protect against such exposures. The failure of DEV to protect against aerosol route infection did not render it detrimental to those who used it; it retained utility because it offered at least some protection against the vast majority of exposures that can occur.

[56] Andrulonis did not suffer harm caused by the use of the Lilly vaccine itself; rather it was his reliance on that vaccine to protect him against aerosol route exposures that contributed to the injuries he suffered. Thus, any culpability on the part of Lilly or Thompson & Sons does not result from the poor "design" of DEV, but rather from the failure to warn others of its limitations. As noted above, even if a product is not defectively designed or manufactured, it can nonetheless be dangerous when put to an intended or

foreseeable use. The "danger" associated with a product may result from misconceptions about the functions that the product can perform on the part of those who use it. If a product is reasonably relied upon to perform a function it is incapable of performing to the detriment of someone foreseeably affected by this reliance, that product can be dangerous.

Employees of Lilly were aware in 1976 and 1977 that the rabies virus can cause disease after airborne route infection, and Lilly had not taken steps to determine whether the rabies vaccine they manufactured was effective in the event of such exposures.[480] Lilly had not modified the package insert included with the rabies vaccine it manufactured to reflect the fact that it was not known whether DEV would provide any measure of protection in the event of aerosol exposures to the virus.[481] The first question presented is whether this failure constituted a breach of Lilly's duty to warn users of potential hazards associated with the use of its vaccine.

[57] The drafters of a package insert must be mindful that "[p]lacing every po-

480. *See* text, *supra* at 1449–1450.

481. Exh. G-125F at 61 (Anderson Deposition); *See* Exh. 87A (Lilly package insert).

tential warning or use requirement onto the label could operate to dilute the most important instructions." *New York State Pesticide Coalition, Inc. v. Jorling*, 704 F.Supp. 26, 29–30 (N.D.N.Y.), *aff'd*, 874 F.2d 115 (2d Cir.1989) (discussing the Environmental Protection Agency's exclusive power to formulate certain labeling requirements). When a vaccine is marketed, it often is not possible to warn of all the potential hazards associated with the use of the vaccine, nor of the dangers associated with imprudent reliance on the vaccine to prevent disease under all circumstances. Aerosol exposure to rabies virus is rare, and it could be argued that in 1976 and 1977 such exposures did not constitute a sufficiently important threat to justify inclusion of warnings directed at them in the DEV package insert. Two important considerations mitigate against this argument, however. First, Lilly affirmatively recommended the use of the vaccine as a pre-exposure preventative measure to spelunkers and laboratory workers, two groups who run an exceptionally higher risk of being exposed to airborne rabies virus.[482] In the United States, there had been three reported deaths of members of those two groups attributed to the airborne transmission of the rabies disease between 1956 and 1976, not an inconsequential number in a period

482. *See* Exh. 87A.

in which total rabies-related deaths in this country was very low. Second, the warnings contained in the DEV package insert were directed at the physicians who administered the vaccine to others,[483] as is commonly the case with warnings accompanying prescription drugs and the like. *See Lindsay v. Ortho Pharmaceutical Corp.*, 637 F.2d 87 (2d Cir.1980). There is evidence that while rabies experts were well aware of the airborne transmission of the virus, this was not well known in the medical community as a whole.[484] Since airborne rabies posed a real and serious threat to those most likely to make pre-exposure use of the Lilly vaccine, and since there was little reason to believe that those administering the vaccine would know of the danger posed by aerosols containing rabies virus, Lilly had a duty to include a warning about the possible ineffectiveness of DEV against the airborne transmission of the disease in its package insert, and that duty was breached.

[58–61] This failure to warn rendered the Lilly vaccine "defective" as far as strict products liability theory is concerned. The court also finds that the NYSDOH scientists were making a foreseeable, even in-

483. Exh. G-125G at 166 (Peck Deposition).

484. *See* Tr. at 799-800 (Tillotson).

tended, use of the vaccine by using it as a means of protection against laboratory exposures to the rabies virus. A closer question is whether Lilly's failure to warn was a proximate cause of the injuries suffered by Andrulonis. The court finds that it was. The vaccine was widely distributed to individuals with varying degrees of knowledge concerning the rabies virus. Thus, it would not have been reasonable for Lilly to assume that users of the vaccine would appreciate the danger at issue in this case, and consequently the "obviousness" exception is inapplicable here. The court has also found that neither Debbie nor Andrulonis had actual knowledge that the vaccine might not provide protection in the event of aerosol exposures, and thus the "knowledgeable user" exception is inapposite. Nor does the court find that the negligence of either Baer or Debbie was so extraordinary as to constitute an "superceding cause" that produced a "different result that could not have been reasonably anticipated." The hazard created by Lilly's failure to warn was an imprudent reliance on the vaccine, and this is precisely what resulted during the course of the Uni-Glatt experiment of March 29, 1977.

[62] Finally, turning to the fourth and fifth elements of a strict products liability case, the court cannot find on the basis of the evidentiary record compiled in this case that the failure of Jerome Andrulonis to discover the "defect" at issue here was the result of his own negligence, nor that he should have otherwise avoided the expo-

sure he experienced through the exercise of ordinary care. The basis for this finding is discussed in greater detail below.[485]

On the basis of the foregoing, the court finds that the actionable conduct of defendants Lilly and Thompson & Sons contributed to the injuries suffered by Andrulonis.

D. *The Culpable Conduct Attributable to the WARF Defendants*

In their amended complaint, plaintiffs alleged that defendants WARF Institute, Inc., Wisconsin Alumni Research Foundation, Inc., Raltech Scientific Services, Inc., and Ralston Purina Company ("the WARF defendants") negligently recommended use of the Uni-Glatt machine for the enteric coating experiments despite the fact that those defendants should have realized the Uni-Glatt was unsafe for the experiments Dr. Debbie contemplated. Plaintiffs settled their claims against the WARF defendants before trial. The Government now seeks to reduce any damages plaintiff Jerome Andrulonis recovers against it by the WARF defendants' equitable share of responsibility for those damages.

A fundamental problem undercutting any theory upon which the liability of the WARF defendants could be based in the case at bar is the failure of the record

485. *See* text, *infra* at 1503-1508.

evidence to establish that the Uni-Glatt machine was inappropriate for the experiments Debbie conducted. As noted above, the expert testimony did not foreclose the possibility that Debbie's enteric coating experiment could have been accomplished safely while using the Wurster air suspension process or the Uni-Glatt machine.[486] Had the machine been placed within an adequate containment system, Andrulonis would not have been exposed to the aerosolized virus. While it is true that such exposure would also have been avoided had the machine been airtight, the evidence did not establish to the court's satisfaction that the fact that the machine leaked meant that it was necessarily unsafe for the use intended by Debbie.

Even if the Uni-Glatt machine was improperly designed for the use Debbie intended because it leaked, the court sees no basis for holding the WARF defendants liable for the injuries suffered by Andrulonis. To find liability on the facts of this case, the court would have to find that WARF had an obligation to independently investigate the safety of using a rabies virus vaccine in the Uni-Glatt, or that WARF's reliance on representations made

486. *See supra* note 421.

by Drs. Baer and Winkler of the CDC concerning the safety of ERA-derived virus strains was unreasonable, or that WARF should have realized that any exposure to a live rabies virus strain posed a threat to those exposed.

[63] Debbie initially contacted a WARF representative seeking information about coating techniques and was told of the Wurster air suspension process. Thereafter, Debbie received a letter from the WARF representative indicating that he would "check with people" at WARF about the safety of using a rabies virus vaccine in the Uni-Glatt while Debbie contacted Baer and Winkler regarding the safety of the virus to be used.[487] There was no evidence introduced establishing that anyone at WARF independently assessed the safety of using ERA-derived strains in the Uni-Glatt experiments. On the basis of the record evidence, it can only be concluded that WARF officials relied on the letters submitted by the scientists from the CDC in concluding that the experiment Debbie proposed could be safely accomplished with the Uni-Glatt. There is no credible evidence, however, that Debbie relied on WARF officials to verify the safety of the Uni-Glatt machine for the experiments he wished to conduct. Therefore, WARF's failure to independently assess the safety of Debbie's research project when conducted with the Uni-Glatt is not actionable.

487. *See* text, *supra* at 1455-1456.

[64] Moreover, the court finds that WARF officials did not act unreasonably in recommending the Uni-Glatt because they relied on letters provided by Baer and Winkler which inadequately addressed the hazards associated with the virus strains that were actually used in the Uni-Glatt experiments. It cannot be questioned that the expertise of Baer and Winkler concerning rabies virus strains far surpassed that of anyone at WARF, and seeking and relying on such expert opinions, far from being negligent, would seem to have been the prudent course for WARF to take.

Finally, there is nothing upon which the court can base a finding that the WARF defendants should have realized the danger inherent in an aerosol exposure to the virus strains used by Debbie. WARF sought information on the safety of the virus strains to be used, and the information provided and reasonably relied upon by WARF would not indicate to a non-expert that humans would be endangered by the aerosols created by the Wurster air suspension process employed by the Uni-Glatt machine. It is true that Harlen Hall, a WARF representative who travelled to Griffin to provide NYSDOH personnel with technical assistance concerning the operation of the Uni-Glatt machine, witnessed a coating run conducted with the machine outside of a physical containment sys-

tem.[488] This fact alone cannot form the basis for finding liability against the WARF defendants absent evidence, not present in the record, that Hall possessed or should have possessed knowledge alerting him to the hazard created by exposure to aerosols containing rabies virus.

In sum, the Government failed to establish that the WARF defendants are liable to plaintiff Jerome Andrulonis for the injuries he suffered.

E. *The Culpable Conduct Attributable to the Glatt Defendants*

[65] Plaintiffs also settled their claims against defendants Glatt GmbH and Glatt Air Techniques, Inc. ("the Glatt defendants") before trial. The Glatt defendants manufactured the Uni-Glatt machine used in the March 29 experiment, and rented that machine to Debbie after a referral from WARF. The Government contends that the Uni-Glatt was defective, and that Andrulonis' damage recovery should be reduced by the percentage of the relative fault of the Glatt defendants.

There is no record evidence upon which a finding against the Glatt defendants on either a negligence or strict products liability theory can be based. The evidence failed to establish that the Uni-Glatt used

488. *See* text, *supra* at 1457-1459.

in the March 29 experiment was defectively constructed. As noted above, the fact that the machine was not airtight does not inevitably mean that it was inappropriate for the purpose Debbie intended. Moreover, there is no evidence that the machine was being put to an intended or foreseeable use, as assessed either from the vantage point of the Glatt defendants (who, unlike the other defendants, apparently had no actual or imputable knowledge of the nature of Debbie's experiments). Nor is there evidence that the ordinary consumer who might rent or purchase such a machine would view the enteric coating experiments as an intended or foreseeable use of the machine. There was no evidence that alternative designs for the Uni-Glatt machine were feasible. To the extent that a failure to warn claim could be based on the Glatt defendants failure to advise consumers that the Uni-Glatt was not airtight, the court notes that this attribute of the machine was known to the NYSDOH employees, including Debbie and Andrulonis, before the accident of March 29, and thus liability cannot be based on this omission by the Glatt defendants. In sum, the Government failed to demonstrate that the culpable conduct of either of the Glatt defendants was a contributing cause of the injuries Andrulonis suffered.

F. *Plaintiff's Culpable Conduct*

[66–69] Under New York law, a plaintiff's recovery of damages for personal injury caused by the culpable conduct of another is to be "diminished in the proportion which the culpable conduct attributable to the [plaintiff] bears to the culpable conduct which caused the damages." N.Y. C.P.L.R. § 1411 (McKinney 1976). The United States argues that Jerome Andrulonis was contributorily negligent in exposing himself to airborne suspensions containing live rabies virus and that Andrulonis "assumed the risk of developing rabies in the course of his activities" at Griffin.[489]

489. Prior to the enactment of New York's comparative negligence statute, N.Y.C.P.L.R. Article 14–A (McKinney 1976), the doctrine of assumption of risk erected an absolute bar to recovery by an injured plaintiff. *Schneider v. Revici*, 817 F.2d 987, 994 (2d Cir.1987) (applying New York law). Assumption of risk was predicated on contract principles rather than a theory of "culpable conduct": the plaintiff either expressly or impliedly agreed to absolve the defendant of responsibility for an injury-causing act, even a negligent one, which was the result of the plaintiff's decision to enter into an activity with actual or imputed knowledge of the activity's danger. *Id.*; *Arbegast v. Board of Education*, 65 N.Y.2d 161, 165, 490 N.Y.S.2d 751, 754–55, 480 N.E.2d 365, 368–69 (1985). The common law distinguished between "express" and "implied" assumption of risk. "Express" assumption of risk occurred when the plaintiff agreed in advance that the defendant need not exercise reasonable care for the plaintiff's benefit and that the defendant would not be liable for the consequence of conduct that would otherwise be

The burden of proving the culpable conduct of a plaintiff is placed on the party seeking to diminish that plaintiff's recovery. N.Y. C.P.L.R § 1412 (McKinney 1976); *see also* N.Y.C.P.L.R. § 3018(b) (McKinney Supp. 1989).

Contributory negligence "is conduct on the part of the plaintiff, contributing as a legal cause to the harm he has suffered, which falls below the standard to which he

deemed negligent. *Arbegast*, 65 N.Y.2d at 169, 490 N.Y.S.2d at 757, 480 N.E.2d at 371. "Implied" assumption of risk was based on the plaintiff's voluntary and unreasonable consent to expose himself to a risk of harm with full understanding of the possible harm to which he was exposing himself. *Id.* Under current New York law, express assumption of risk remains an absolute defense to an action in tort, whereas implied assumption of risk has been subsumed within the "culpable conduct" language of N.Y. C.P.L.R. § 1411 and merely diminishes the amount a plaintiff can recover. *Id.* at 170, 490 N.Y.S.2d at 757-58, 480 N.E.2d at 371-72; *Schneider*, 817 F.2d at 995. The Government did not distinguish between express and implied assumption of risk in its post-trial submissions to the court. The record is devoid of evidence that Jerome Andrulonis expressly agreed to relieve Dr. Baer of his duty to exercise reasonable care, however, and thus the court will confine its discussion in the text to a theory based on Andrulonis' implied assumption of risk.

is required to conform for his own protection." Prosser & Keeton § 65, at 451; *O'Connor v. G & R Packing Co.*, 74 A.D.2d 37, 49, 426 N.Y.S.2d 557, 565 (2d Dept.1980), *aff'd*, 53 N.Y.2d 278, 440 N.Y.S.2d 920, 423 N.E.2d 397 (1981). At issue is "whether the plaintiff's conduct conformed to the standard which an ordinarily prudent person would have observed under the circumstances." *O'Connor*, 74 A.D.2d at 49, 426 N.Y.S.2d at 565. A plaintiff can be said to have assumed the risk of incurring the injury he ultimately suffered if he "voluntarily encounter[ed] the risk of harm from [the] defendant's conduct with full understanding of the possible harm to himself...." *Arbegast v. Board of Education*, 65 N.Y.2d 161, 169, 490 N.Y.S.2d 751, 757, 480 N.E.2d 365, 371 (1985). The evidence at trial clearly established that Andrulonis, like Debbie, was aware that the Uni-Glatt machine was not airtight and that the machine was not operated within a physical containment system during any of the experiments in which it was used at Griffin. Further, it is clear that Andrulonis was aware that aerosols containing rabies virus were created during the course of the Uni-Glatt experiments. Whether Andrulonis was contributorily negligent in exposing himself to aerosols containing rabies virus on March 29, 1977 turns on the nature and degree of Andrulonis' actual or imputed knowledge concerning the airborne transmission of the rabies virus, the risk associated with the virus strain used in the March 29 experiment, and the principles of risk assessment.

In determining whether Jerome Andrulonis was contributorily negligent in aiding Debbie during the March 29, 1977 Uni-Glatt experiment, two lines of inquiry must be pursued. First, the court must determine the degree to which an individual performing the functions Andrulonis performed in the course of his employment would reasonably be required to be familiar with the scientific knowledge discussed in this memorandum-decision regarding the rabies virus and principles of laboratory safety. This is the purely objective part of the court's inquiry: if the position Andrulonis held with NYSDOH required him to have knowledge that should have alerted him to the dangers involved in the manner in which the Uni-Glatt experiment of March 29 was being conducted and he nonetheless exposed himself to those dangers, he failed to conform to the standard of reasonableness imposed by New York law, regardless of whether Andrulonis *actually* possessed the knowledge in question. If this knowledge is not properly chargeable to Andrulonis by virtue of the position he held, the court must attempt to determine Andrulonis' actual knowledge in order to decide whether he reasonably should have been aware that he was placing himself at great risk in carrying out the Uni-Glatt experiment in the manner it was conducted.

A vast, generally comprehensive record has been compiled by the attorneys representing the parties involved in this case.

Yet, there is virtually nothing in the record concerning the minimum degree of knowledge expected of laboratory technicians responsible for the tasks Andrulonis performed by the scientific community as a whole. Moreover, surprisingly little evidence has been presented regarding the extent of Jerome Andrulonis' actual knowledge of the rabies virus or of laboratory safety principles.

In March 1977, Jerome Andrulonis held the title of "Senior Bacteriologist (Virology)." [490] The New York State Department of Civil Service required that someone holding that position possessed a bachelor's degree with a background in biology and chemistry, and had served as a bacteriologist with the State for at least one year.[491] A Senior Bacteriologist (Virology) was charged with "direct[ing] the examinations performed to aid in the diagnosis of viral and rickettsial infections." [492] This work

490. Tr. at 95–96 (Debbie).

491. Exh. 4 (New York State Department of Civil Service, "Specifications for Bacteriologist Series" (revised March 1965)).

492. Exh. 4.

included the performance of diagnostic tests, the cultivation, isolation, and identification of various viral and rickettsial strains from materials submitted by local physicians and other laboratories for diagnosis, the supervision of the performance of serological tests, the supervision of procedures used to isolate viral and rickettsial strains from animals, and the instruction of new and visiting laboratory personnel in laboratory procedures and "precautions necessary to prevent laboratory infection." [493]

The tasks Andrulonis performed at Griffin were those assigned to him by Dr. Debbie or Debbie's superiors.[494] Andrulonis' work was primarily diagnostic; he and co-worker Charles Trimarchi performed the bulk of the diagnostic work at Griffin's rabies laboratory.[495] Andrulonis was responsible for the performance of titrations. He also prepared suspensions containing rabies virus, performed procedures associated with the passage of virus in cell culture systems, diagnosed rabies in animals submitted to Griffin by individuals or other laboratories, and immunized animals.[496]

493. Exh. 4.

494. Tr. at 486 (Debbie).

495. Tr. at 94–95, 253 (Debbie).

496. Tr. at 94–95, 253, 318 (Debbie).

Andrulonis played a leading role in two research projects conducted at Griffin during his tenure with NYSDOH. One project involved the development of an alternative method of determining the titer of a rabies virus strain using tissue culture rather than live mice. The other involved the study of the pathogenesis of the rabies disease in cattle.[497] Andrulonis also assisted Debbie in much of the research the latter directed concerning the development of a method to vaccinate wildlife, and, of course, assisted during the Uni-Glatt experiments.[498]

There is nothing in Andrulonis' job description or in the nature of the tasks he actually performed at Griffin that would require the specialized knowledge of a member of the "community of rabies experts" concerning either the nature of the rabies virus or the less common routes of infection through which the virus might invade a host, such as the airborne route. There is no evidence that his job required him to understand the pathogenesis of the virus after infection through the airborne route, the changes in a rabies virus strain caused by passaging in different cell sys-

497. Tr. at 254–55 (Debbie). There is no evidence that the cattle were subjected to airborne exposures to the rabies virus during these experiments.

498. Tr. at 327–28, 180, 184–88 (Debbie).

tems, or the effectiveness or lack of effectiveness of the Lilly vaccine in preventing the development of disease after exposure through non-bite and non-parenteral routes. The evidence indicates that Andrulonis performed the procedures that had been developed by rabies experts of the past, but not that he possessed the theoretical knowledge underlying the development of those procedures.[499] Nor is the court aware of any testimony or other evidence concerning the minimal level of knowledge of the matters listed above that would be required of a laboratory technician performing the tasks assigned to Andrulonis by NYSDOH, or expected of such a technician by the scientific community in general. Rather, the evidence introduced at trial focused on the level of knowledge reasonably expected of someone within the community of rabies experts or performing and supervising experiments of the sort that should only be conducted by such experts. The court has found nothing in the record remotely suggesting that Jerome Andrulonis himself was a member of the community of rabies

499. *Compare* Exh. G-125A at 139–40 (Rudd Deposition). In 1983, Robert J. Rudd held the position of Senior Bacteriologist at Griffin, and his responsibilities were limited to the diagnosis of rabies in animals and a small amount of research work directed at improving diagnostic procedures.

experts or alternatively that the functions he performed were of the sort properly executed by someone within this limited class.

There is also a troublesome lack of evidence regarding the minimum level of knowledge concerning laboratory safety that was expected of individuals holding positions similar to that of Jerome Andrulonis in 1977. There is no indication that Andrulonis was required to receive instruction regarding laboratory risk assessment as a prerequisite to holding the position of Senior Bacteriologist (Virology) with NYSDOH. The testimony at trial focused on the safety principles that should be known to biosafety specialists and scientists supervising experimental studies in the laboratory. The court has not been alerted to testimony or other evidence in the record establishing whether knowledge of these principles is properly chargeable to Jerome Andrulonis by virtue of the responsibilities he discharged in the course of his work. The job specifications for the position Andrulonis held did require him to instruct new laboratory personnel in the "precautions necessary to prevent laboratory infection," but from the context of the job description it appears that this passage refers to the established methods of avoiding infection during routine diagnostic work. There is no indication that an individual holding the position of Senior Bacteriologist (Virology) with NYSDOH in 1977 was

qualified to independently *establish* what safety measure should be taken with regard to a particular diagnostic activity—that is, to make the risk assessment analysis through which particular safety procedures are formulated. Similarly, there is no indication that a Senior Bacteriologist was qualified to conduct a risk assessment analysis regarding new and untried experimental techniques, a category that would include the use of the rabies virus in the Uni-Glatt experiments conducted at Griffin.

Given the absence of evidence indicating that Andrulonis can be imputed with the degree of knowledge of the rabies virus and principles of laboratory safety that should reasonably have alerted him to the nature of the risk he encountered during the March 29, 1977 Uni-Glatt experiment, the court must look to the evidence presented concerning his actual knowledge of these matters. Andrulonis held a bachelor's degree in biology from Marist College and a master's degree in biology from the College of St. Rose.[500] It appears that his post-graduate studies focused on the rabies virus. In partial fulfillment of the

500. Tr. 1716–18 (Joanna Andrulonis).

requirements for his master of science degree, Andrulonis submitted a graduate dissertation concerning rabies to the faculty of St. Rose. This dissertation, dated May 1973, discussed a study Andrulonis had conducted which involved the growth of the ERA vaccine in a tissue culture derived from the kidneys of pigs (PK/15). Andrulonis gave a general overview of the rabies virus and the history of the development of rabies vaccines before describing his own study and the results he obtained.[501] Specifically, he discussed the development of the ERA vaccine, and the vaccine's "lack of pathogenicity" for the various animal species "it immunize[s]." [502]

This dissertation, which is the primary evidence of Andrulonis' actual knowledge of the rabies virus placed in the record, does not reveal that Andrulonis knew of the pathogenesis of airborne rabies infection, or even that the rabies virus could be transmitted through the airborne route. Nor does the dissertation indicate that Andrulonis possessed knowledge that should have put him on notice that DEV might not be effective against an aerosol exposure to the rabies virus. In his preliminary discus-

501. *See* Andrulonis Dissertation, *supra* note 10, at 1–12.

502. Andrulonis Dissertation, *supra* note 10, at 11–12, 30–31.

sion. Andrulonis stated that "[a] person or animal is exposed to rabies by the bite of another animal having the virus in its saliva ... [or] if no bite occurs, [by] infected saliva contacting an open wound...."[503] He did not mention that the rabies virus can be transmitted through the air. Later, in his discussion of the results of the study he had conducted with the ERA-PK/15 strain he had produced, Andrulonis noted that the pathogenicity of the ERA vaccine could differ depending on whether it was injected intramuscularly or intracerebrally. Again, Andrulonis failed to acknowledge the airborne route of exposure.[504] Andrulonis discussed the pathogenesis of the disease of rabies following introduction of the virus into a wound through the saliva of an infected animal, but he did not discuss the pathogenesis of the disease following airborne exposure, even though literature indicating the role of the olfactory nerves was in existence at the time Andrulonis wrote his dissertation.[505] Andrulonis brief-

503. Andrulonis Dissertation, *supra* note 10, at 3.

504. Andrulonis Dissertation, *supra* note 10, at 30–31.

505. Andrulonis Dissertation, *supra* note 10, at 3–6; *see* text, *supra* at 46–50, and accompanying notes.

ly reviewed the history of rabies vaccines, but did not reveal an understanding that the antibody response triggered by these vaccines might be ineffective to protect against a non-parenteral exposure to the rabies virus in the laboratory.[506] Finally, the dissertation does not discuss the unpredictable evolutionary mutations a virus undergoes during passaging, nor the idea that a virus strain can become attenuated for one animal species if it is adapted to the cell system of another.[507] In sum, the dissertation itself does not reveal that Andrulonis possessed actual knowledge of studies and principles that should have alerted him to the dangers inherent in the Uni-Glatt experiments.

There is other evidence of Andrulonis' actual knowledge of the rabies virus in the record. In his deposition testimony, Dr. Abelseth stated that it was widely known that an increase in the titer of a rabies virus strain was ordinarily associated with an increase in its pathogenicity, and Andrulonis' dissertation indicates that Andrulonis was aware of this fact.[508] Dr. Debbie testi-

506. Andrulonis Dissertation, *supra* note 10, at 8–12; *see* text, *supra* at 49–50, and accompanying notes.

507. *See* text, *supra* at 1439–1441.

508. Tr. at 617 (Abelseth Deposition); *see generally* Andrulonis Dissertation, *supra* note 10.

fied that Andrulonis knew that two individuals exposed to the atmosphere of Frio Cave in the 1950s may have contracted rabies through the airborne route, and that Andrulonis was aware of the veterinarian who apparently contracted the disease after an aerosol exposure in the laboratory in Texas in 1973.[509] Charles Trimarchi attested that he had discussed the airborne transmission of the rabies virus with many individuals, including Andrulonis, although the substance of these conversations was not revealed in Trimarchi's deposition.[510] Trimarchi also indicated that both he and Andrulonis made periodic presentations to public health workers in the State of New York, and in those presentations they would mention that the virus could be transmitted through the airborne route.[511] Again, the substantive content of the infor-

509. Tr. at 399 (Debbie); *see* text, *supra* at 1445 and 1447.

510. Exh. G-125C at 122-23, 304-05, 326, 678 (Trimarchi Deposition).

511. *See* Exh. G-125C at 652-60, 305-07 (Trimarchi Deposition).

mation Andrulonis related in these presentations was not described.[512]

[70] Thus, the Government established at trial that Andrulonis knew that the disease of rabies can be acquired after an airborne exposure, that Andrulonis was familiar with the leading cases in which it was suspected that humans had died as a result of aerosol exposures to the rabies virus, and that Andrulonis was aware that the danger associated with a particular virus strain increased as the titer of that strain increased. It was not established that Andrulonis knew anything about the pathogenesis of the disease of rabies after an aerosol exposure, understood the "theory" underlying the way in which rabies

512. Trimarchi's deposition testimony also revealed that a file containing copies of articles relating to rabies was maintained at Griffin and that those articles were often circulated among the workers in the rabies laboratory. Exh. G–125C at 328 (Trimarchi Deposition). Surprisingly, it was not established that any of the articles cited thus far in this memorandum-decision were circulated at Griffin, whether Andrulonis actually read them, or whether Andrulonis should have read them as a part of his duties at NYSDOH. The court would guess that Andrulonis did read this literature, but cannot base a finding on guesses. The record is insufficiently developed to allow the court to do anything but speculate on this issue.

vaccines generally or DEV in particular protected an individual exposed to the rabies virus, or appreciated the fact that a virus strain that was "attenuated" for one species could be lethal to another species, including man. It was also not established that Andrulonis had any actual knowledge of laboratory safety principles of risk assessment, or that he appreciated that additional safeguards not taken during the Uni-Glatt experiments were available. The court finds that knowledge of these latter principles (whether actual or imputed) not only is a prerequisite to a finding of negligence on the part of Drs. Baer and Debbie,[513] it is indispensable to a finding of contributory negligence on the part of Andrulonis. Accordingly, the court cannot conclude that the Government has demonstrated by a fair preponderance of the evidence that Andrulonis was aware of facts and principles that should have alerted him that the Uni-Glatt experiments were not being performed in a reasonably safe manner.

The court's findings in support of its conclusion that the Government failed to meet its burden of proving that Andrulonis was contributorily negligent also dispose of

513. *See* text, *supra* at 1476–1477 and 1490.

the claim that Andrulonis' recovery should be diminished under the doctrine of assumption of risk. It is true that any laboratory worker who is exposed to the rabies virus in his work knows that it is possible to contract the disease of rabies as a result, just as a driver of an automobile knows it is possible he may have an accident by travelling on a highway. There is no evidence, however, that Andrulonis appreciated the markedly increased risks associated with the March 29, 1977 Uni-Glatt experiment. Absent a showing that Andrulonis possessed knowledge that would have allowed him to fully comprehend the risk associated with that experiment, the court cannot find that he assumed the risk of harm created by the actions and omissions of Drs. Baer and Debbie.

G. *Apportionment of Liability*

[71] Where, as here, two or more tortfeasors independently breach duties owed to an injured party and each tortfeasor's negligence is a substantial cause of the ultimate injury suffered, those tortfeasors can claim contribution from one another in accordance with the fact-finder's apportionment of fault. N.Y.C.P.L.R. §§ 1401-1404 (McKinney 1976). The court's assessment of the degree of responsibility borne by each of the defendants whose culpable conduct contributed to the injuries suffered by Jerome Andrulonis will largely reflect its impressions of the evidence summarized above, and that evidence will not be re-

peated here. Certain considerations do merit mention, however.

First, although Dr. Baer realized that the Uni-Glatt machine was not an airtight, closed system before the ERA-BHK/21 virus strain was introduced into the machine, the court is cognizant of the fact that individuals do not as fully appreciate the implications of the facts they observe at the time they observe them as they do after a time of reflection. Baer became aware that the Uni-Glatt was not airtight and was not being operated within an appropriate containment system on the morning of March 29, 1977. Dr. Debbie, in contrast, had conducted experiments with the Uni-Glatt on three prior occasions over a period of a year. His failure to appreciate the hazard to which he was exposing his workers seems more unreasonable than Baer's failure to recognize the same hazard. Moreover, while Baer should have realized that the Uni-Glatt machine leaked, Debbie knew that the machine leaked *heavily*, for he had witnessed the substantial escape of red dust from the machine in prior coating runs. Baer did not witness a visible leakage of dust until after the ERA-BHK/21 strain had been sprayed onto the nonpareils within the machine's transparent column.

On the other hand, Baer's knowledge of the rabies virus was far superior to that of Debbie. Debbie has been imputed with knowledge of the principles and studies that have been discussed in this opinion; in

March 1977, Baer actually possessed that knowledge. Importantly, he had knowledge of the facts indicating that airborne rabies virus probably infected the olfactory nerves within the nasal cavity and that the Lilly vaccine might not offer any protection in the event of such an infection. This factor would favor the placement of a greater share of the blame for the accident that occurred on March 29 on Dr. Baer.

Finally, the court finds that the inadequacy of Lilly's package insert is clearly a secondary causal factor of Andrulonis' injuries. Baer and Debbie had access to the same information available to Lilly, and both appear to have possessed greater ability to comprehend the implications of that information than anyone employed by Lilly. More importantly, Lilly had no connection with two of the most important causal factors contributing to this tragedy: the use of a highly concentrated virus strain of unknown virulence in an experiment that created aerosols, and the failure of the scientists involved to provide appropriate containment for those aerosols.

[72] In sum, the court concludes that as between the United States, NYSDOH, Lilly, and Thompson & Sons, damages should be apportioned as follows:

The United States	30%
NYSDOH	65%
Lilly/Thompson & Sons	5%

V. DAMAGES

To evaluate the loss Jerome Andrulonis has suffered as a result of the negligence that has been found in this case, the court must survey the nature of his existence before he contracted the disease of rabies and examine the changes in his life caused by that disease. By summarizing the details of a life, however, a court cannot hope to capture its essence. To borrow the words of the historian Jakob Burkhardt, such details reveal only "the underside of the tapestry"—the knots and stitches that give a life its contours, but not the shadings and accents that give it identity. Conscious of its limitations, the court will outline what it knows of Jerome Andrulonis both before and after the onset of his illness.

A. *Background*

Jerome Andrulonis met his wife, Joanna, at a college mixer in 1964. They married three years later, shortly after her graduation. By that time, Jerome had obtained a bachelor's degree in biology from Marist College, and he taught science and biology at a high school on Long Island, New York from 1966 until 1968. In May, 1968, Joanna gave birth to the couple's first child, a girl they named Anne. Jerome, not happy teaching high school science, took and passed New York State's Professional Careers Test that same month, and afterwards was offered a number of positions

with the State. He was interested in doing research work and accepted a position at Griffin Laboratory. In August 1968, the Andrulonis family moved to the Albany area.[514]

Jerome began his career with the State with an entry level position in the "Bacteriologist Series" as defined by the State's Department of Civil Service.[515] As a bacteriologist (pay grade 14) Andrulonis performed assigned portions of research projects and otherwise assisted higher ranking bacteriologists and scientists at Griffin. With the supervision of superiors, Andrulonis directed the serologic, cultural, and microscopic examination of specimens through which any pathogenic organisms present in those specimens can be identified.[516] As he worked full-time at Griffin, Andrulonis pursued an advanced degree in biology at the College of St. Rose in Albany. Two more children were born to the Andrulonises, Bucky in August, 1969 and Joanna, Jr. in the Spring of 1971; Jerome

514. Tr. at 1716–19 (Joanna Andrulonis).

515. Tr. at 1717 (Joanna Andrulonis); *see* Exh. 4 (New York State Department of Civil Service, "Specifications for Bacteriologist Series" (revised March 1965)).

516. Exh. 4.

obtained his master's degree from St. Rose in 1973; he was promoted to the position of Senior Bacteriologist (pay grade 18) at Griffin.[517] He enjoyed his job at Griffin, and wanted to continue in his work with the rabies virus. At the time he became ill, he was studying for the civil service examination for the position of Associate Bacteriologist (pay grade 23), the highest position in the State's Bacteriologist Series.[518]

Before his illness, Jerome Andrulonis was an active, athletic man. He was an avid sports fisherman; he regularly played golf and tennis; he enjoyed engaging in recreational activities with his family. He and Joanna were active in church and community affairs. Jerome coordinated his parish's junior high school religious education program, and with Joanna taught religious education and conducted marriage encounter programs.[519] Joanna Andrulonis described her husband as a gentle, modest man with many friends.[520] Perhaps the most revealing testament of the character of Jerome Andrulonis before his illness is the dedication of his wife and family in caring for him through the years that followed it.

517. Tr. at 1717–19 (Joanna Andrulonis).

518. Tr. at 1762 (Joanna Andrulonis); Exh. 4.

519. Tr. at 1720–22 (Joanna Andrulonis); *see also* Tr. at 1709 (Anne Andrulonis).

520. Tr. at 1719 (Joanna Andrulonis).

Andrulonis was thirty-four years old and in good health when he experienced a massive exposure to aerosolized rabies virus on March 29, 1977. As a result of this exposure, he contracted rabies encephalitis. The anatomical damage, pain and suffering, and loss of enjoyment of life's pleasures described here, along with the economic losses attendant to those injuries, are causally related to the rabies encephalitis Andrulonis contracted.[521]

The onset of Andrulonis' illness, his hospitalization, and his decline into a comatose state have already been detailed.[522] Briefly, Andrulonis initially suffered non-specific flu-like symptoms such as fever and malaise; subsequently he became extremely agitated and delusional; he then became more lethargic and on April 21, 1977 he slipped into a coma. His vital signs were unstable and he suffered severe muscle spasms that required the administration of medication. He was placed on full respiratory support, a tracheostomy was performed to assist breathing, and the difficul-

521. Tr. at 729-30 (Tillotson); Tr. at 859-60 (Rodichok) (anatomical damage caused by viral encephalitis); Tr. at 1529 (Testimony of Dr. Anthony V. DeTommasi) (plaintiff's dementia is secondary to rabies encephalitis).

522. *See* text, *supra* at 1465-1467.

ties Andrulonis had in swallowing required the performance of a gastrostomy on May 27.

Toward the end of May, Andrulonis began to show signs that he was emerging from his coma. On May 23, for example, Andrulonis responded to some simple commands.[523] In the first part of June, he was propped in a chair and his eyes remained open for a short time.[524] Gradually, Andrulonis became more alert and was able to move around somewhat on his own, although he continued to have problems relating to his environment.[525] As Andrulonis became more aware of what was happening around him, however, he became more agitated and combative.[526] He resisted treatment and fought off the doctors and nurses who attempted to examine him, requiring hospital personnel to restrain him in his bed or in his chair through the use of a posey, a jacket with straps that allows a patient the freedom to move his arms and legs but prevents him from moving

523. Tr. at 694–95 (Tillotson).

524. Tr. at 1725 (Joanna Andrulonis).

525. Tr. at 701–02 (Tillotson).

526. Tr. at 702 (Tillotson); Tr. at 1727 (Joanna Andrulonis).

about.[527] Toward the end of June, Andrulonis was placed on Valium in an attempt to make him more easily manageable, and in July a tranquilizer, Mellaril, was prescribed. Andrulonis continued to receive medication intended to control his muscle spasms and his high blood pressure.[528]

In August 1977, a speech language pathologist began treating Andrulonis.[529] Upon first encountering him, the pathologist found her patient to be rigid and totally speechless. His eyes were open, but he did not appear to be cognizant of his surroundings. After two weeks of therapy, the pathologist was able to establish eye contact with Andrulonis; four to six weeks later, Andrulonis uttered his first sound since he slipped into his coma.[530] At this point, Andrulonis' ability to perform higher intellectual functions was severely limited; his treating physician testified that at best Andrulonis had the intellectual function of a two-year-old infant.[531]

527. Tr. at 702–03, 710–11 (Tillotson); Tr. at 1727 (Joanna Andrulonis).

528. Tr. at 704 (Tillotson).

529. Tr. at 1576 (Testimony of Rebecca Guerrette Rosen).

530. Tr. at 1577–78 (Rosen).

531. Tr. at 709 (Tillotson).

In September 1977 Andrulonis began displaying some of the symptoms that characterized the latter stages of his initial hospitalization at AMCH. Andrulonis exhibited agitation not only by thrashing out violently and unpredictably but also by engaging in nondirected repetitive movements. For example, if left seated unattended, Andrulonis would constantly move his feet beneath him as if he were walking.[532] He went through periods in which he would repeatedly grind his teeth together, necessitating at one point consultation with a dentist, who contemplated the construction of a protective device to prevent the patient from wearing his teeth down.[533]

From the outset of Andrulonis' hospitalization, EEGs revealed very abnormal slow brain electrical activity, evidencing a substantial loss of brain function and suggesting some form of widespread anatomical damage to the brain.[534] Any doubt that Andrulonis' illness had caused permanent anatomical damage was removed by a CAT

532. Tr. at 711 (Tillotson); Exh. 123 (AMCH Progress Notes—History Sheet dated September 24, 1977).

533. Tr. at 712 (Tillotson); Exh. 123 (AMCH Progress Notes).

534. Tr. at 705–07 (Tillotson); Tr. at 839–40 (Rodichok).

scan taken October 20, 1977. That CAT scan revealed that Andrulonis had suffered a devastating loss of brain matter.[535] The October 20 CAT scan, as well as others taken subsequently, showed that Andrulonis lost a substantial amount of tissue in the frontal and temporal lobes of the cortex as a result of his illness. The frontal and temporal lobes constitute that portion of the brain that ordinarily is responsible for most advanced intellectual functions and determines behavior and personality. This cortical atrophy was accompanied by an expansion of the cerebral ventricles and an excessive accumulation of fluid in the spaces within Andrulonis' brain where tissue previously existed.[536] It is theorized that this anatomical damage was caused by the reaction of Andrulonis' body in attempting to fight off his viral infection.[537]

535. Exhs. 131 and 132 (Enlarged reproductions of October 20, 1977 Cat scan); Tr. at 707–08 (Tillotson); Tr. at 845–46 (Rodichok).

536. Tr. at 845–49 (Rodichok); Tr. at 708 (Tillotson); *compare* Exhs. 128, 129, and 130 (Enlarged reproductions of normal CAT scans taken April 22, 1977) with Exhs. 131, 132, 133, and 134 (Enlarged reproductions of CAT scans taken October 20, 1977, January 12, 1978, and August 8, 1979).

537. Tr. at 717–18 (Tillotson).

In October 1977, Andrulonis underwent a second gastrostomy. This surgical procedure was required because the gastrostomy tube that had been inserted into Andrulonis' stomach either fell out or had been pulled out and the surgical site had completely sealed over.[538] That same month, Andrulonis suffered a recurrence of a urinary tract infection. The patient became increasingly combative, and his prescription for tranquilizers was altered.[539]

By this time, Andrulonis was allowed to leave the hospital and visit his home approximately once a week. Andrulonis had been permitted to leave the hospital for the first time a couple of months earlier, on August 17, 1977, to attend his son's birthday party.[540] During these visits, Andrulonis was accompanied by registered nurses from the hospital, and was transported by wheelchair. He was unable to tend to his personal hygiene. Andrulonis continued to take most of his nourishment through the gastrostomy tube that had been inserted through his stomach, even

538. Tr. at 714 (Tillotson); Exh. 123 (AMCH Progress notes—History Sheet dated October 2, 1977).

539. Tr. at 714–16 (Tillotson).

540. Tr. at 710 (Tillotson); Tr. at 1727 (Joanna Andrulonis).

when he was visiting his home.[541] Joanna Andrulonis consistently reported that her husband's behavior at home was markedly better than it was in the hospital, but it was suspected that these reports were colored by Joanna's unrealistic optimism concerning her husband's prospects for recovery.[542] In any event, the duration of Andrulonis' weekly visits home gradually increased over time.

On March 9, 1978, eleven months after the initial on-set of symptoms, Andrulonis was released from AMCH. By this time, Andrulonis could respond to very simple commands, but he had virtually no attention span. He often seemed preoccupied and was not interested in participating in simple activities.[543] His behavior continued to be aggressive and somewhat combative, despite the considerable amount of medication he regularly received.[544] At the time of his discharge, Andrulonis was unable to bathe himself or dress himself, and he was incontinent.[545] Andrulonis was able to feed himself with difficulty, provided

541. Tr. at 1728–29 (Joanna Andrulonis); Tr. at 719–20 (Tillotson).

542. Tr. at 720 (Tillotson).

543. Tr. at 721 (Tillotson); Tr. at 1732 (Joanna Andrulonis).

544. Tr. at 722 (Tillotson).

545. Tr. at 1732 (Joanna Andrulonis).

that his family or an attendant cut his food for him. He tended to eat voraciously, packing his mouth so full of food that he could not chew it, and instead would try to swallow his food whole. Thus, it was necessary for a family member or nurse to monitor his eating constantly.[546]

Shortly after leaving the hospital and returning home, Andrulonis became ambulatory. Soon thereafter, he developed a pacing syndrome, and if left by himself Andrulonis would walk and pace constantly.[547] Andrulonis had difficulty controlling the muscles of his mouth, his tongue writhed, and he drooled excessively. Besides disturbing those who cared for him, this condition seriously interfered with Andrulonis' speech therapy.[548] Andrulonis frequently appeared quite agitated. He would sit for brief periods and rock back and forth, and then would rise and pace rigidly.[549] Andrulonis' activity was nondirected, and Andrulonis was frequently oblivious to those around him. None-

546. Tr. at 1732 (Joanna Andrulonis); Tr. at 1354 (Testimony of Kenneth H. Piedmont).

547. Tr. at 1733 (Joanna Andrulonis); *see* Tr. at 711 (Tillotson)

548. Tr. at 1582 (Rosen).

549. Tr. at 1353 (Piedmont).

theless, in the months following his discharge from AMCH, Andrulonis was somewhat manageable.

He did not remain so. While always exhibiting signs of agitation, after the Winter of 1979 Andrulonis became frenzied and uncontrollable. He was restless and extremely excited, pacing furiously day and night.[550] Andrulonis' restlessness was so extreme that he would sleep only one hour in a twenty-four hour period, pacing constantly during the hours he was awake. Administration of Thorazine, a tranquilizer, and Tegretol, an anticonvulsant and pain reliever, failed to calm Andrulonis' agitation.[551] He became completely uncommunicative, and he was prone to violent outbursts of anger.[552] His activity became so frenzied at one point that he lost between twenty and thirty pounds within a one month period.[553] Finally, on April 30, 1979, after a particularly destructive outburst in the Andrulonis home, Jerome Andrulonis was admitted to the Albany Medical Center

550. Tr. at 1521 (DeTommasi); Tr. at 1359 (Piedmont).

551. Tr. at 1522 (DeTommasi); Tr. at 1733 (Joanna Andrulonis).

552. Tr. at 1521-22 (DeTommasi).

553. Tr. at 1733 (Joanna Andrulonis); Tr. at 1359 (Piedmont).

Hospital's psychiatric unit.[554] He was held on the locked psychiatric ward at AMCH for approximately four months.[555]

During the course of this second hospitalization, doctors prescribed a variety of psychotropic medications in an attempt to control Andrulonis' extreme agitation, without success.[556] His private nurses continued to care for him at the hospital, since Andrulonis did not (and to this day does not) adapt well to change and required some continuity of care while in the hospital.[557] Whatever reassurance this continuity provided, however, was insufficient to calm Andrulonis' aggression. Andrulonis lashed out against people and ran wildly through the psychiatric ward, brushing others aside or running over them. Andrulonis' running was so relentless and unrestrained that he developed shin splints, cracked heels, and callouses.[558] He experienced dramatic emotional mood shifts,

554. Tr. at 1360 (Piedmont).

555. Tr. at 1735 (Joanna Andrulonis); Tr. at 1360 (Piedmont).

556. Tr. at 1522–23 (DeTommasi).

557. Tr. at 1360–61 (Piedmont); Tr. at 1738–39 (Joanna Andrulonis).

558. Tr. at 1368–69 (Piedmont); Exh. 124 (AMCH Progress Notes).

laughing uncontrollably one moment and crying the next.[559] For most of his stay in AMCH's psychiatric ward, Andrulonis' hyperactivity was so severe that it was necessary to restrain him with poseys and arm and leg restraints.[560]

Toward the end of this second hospitalization, doctors at AMCH prescribed Prolixin, a tranquilizer used in the treatment of the manifestations of psychotic disorders. This drug stabilized Andrulonis' behavior to a degree sufficient to allow his family to take him back home.[561] On August 5, 1979, more than three months after his admission to the psychiatric ward, Andrulonis was again discharged from AMCH. While still predisposed toward hyperactivity, Andrulonis was calmer when administered Prolixin, even showing signs of drug-induced lethargy on occasion.[562]

After his second hospitalization, Andrulonis could not name simple objects nor consistently identify his wife or children. He still had frequent periods of agitation in which he would talk to himself and rock

559. Tr. at 1368 (Piedmont).

560. Tr. at 1364–65 (Piedmont).

561. Tr. at 1523–24 (DeTommasi); Tr. at 1737 (Joanna Andrulonis).

562. Tr. at 1372–73 (Piedmont); Tr. at 1737 (Joanna Andrulonis); Tr. at 1525 (DeTommasi).

back and forth in his chair, unable to sit still. His insomnia persisted.[563] As the end of 1980 neared, Andrulonis' episodes of hyperactivity and his propensity toward violent outbursts again worsened. He became impatient easily, and expressed this impatience by throwing household objects or by lashing out against his family. A large and strong man who was very active in athletics before the onset of his illness, Andrulonis was very difficult to control during his violent episodes. He had the strength to hurl chairs and tables across a room, and during his most violent episodes he caused a substantial amount of damage to the Andrulonis household.[564] Andrulonis would punch, kick, grab, twist or pull the hair of his wife and children.[565] He would lash out and strike the nurses aides who attended him, as well as the therapists who counseled him.[566] He would kick the family dog; he would become angry and chase his children through the house.[567] Most

563. Tr. at 1526 (DeTommasi).

564. Tr. at 1741–42 (Joanna Andrulonis); *see also* Tr. at 1584–85, 1587 (Rosen).

565. Tr. at 1586 (Rosen).

566. Tr. at 1593 (Rosen); Tr. at 1743 (Joanna Andrulonis).

567. Tr. at 1587, 1592–93 (Rosen).

often, Andrulonis' rage would be directed at the person who has remained closest to him in the aftermath of his debilitating illness, his wife Joanna. Subject to dramatic mood shifts, Andrulonis could be hugging or kissing his wife one moment and be swearing at her, kicking her, or punching her the next.[568]

The daily dosage of Prolixin that had been prescribed for Andrulonis, which was high when Andrulonis was discharged from AMCH in August 1979, was increased further around December 1980, without much discernable improvement in his tormented behavior. In January 1981, the psychiatrist treating Andrulonis prescribed the anti-psychotic drug Loxitane in place of the Prolixin he was being administered, but Andrulonis' agitation persisted. In August of that year, Andrulonis struck his wife in the face during a visit to his psychiatrist's office and acted erratically. In an October visit to the same doctor, Andrulonis seemed more angry and restless.[569]

568. Tr. at 1593 (Rosen); Tr. at 1742 (Joanna Andrulonis).

569. Tr. at 1526–27 (DeTommasi).

Andrulonis seemed in constant turmoil, as evidenced by his frenzied behavior and the increased frequency of his violent outbursts. In 1981, his neurologist prescribed Tegretol, an anticonvulsant drug, and for a time his violence subsided. Andrulonis continued to be administered Loxitane and Tegretol until September 1984, when the Tegretol was discontinued after Joanna Andrulonis complained that the drug seemed to make her husband's violent outbursts worse. Since the Tegretol has been discontinued, Andrulonis' agitation and periods of violence have persisted, but his outbursts are significantly less frequent than they were between 1981 and 1984, and he now has longer periods of stability.[570]

Throughout this decade, Andrulonis has continued to show evidence of severe brain damage. Andrulonis' language skills remain very limited. Upon his initial release from AMCH in March 1978, Andrulonis was able to utter a handful of words inconsistently, usually repeating what he heard.[571] The speech language pathologist who has treated Andrulonis since his discharge testified that after that initial discharge, it was difficult just to capture her

570. Tr. at 1754–55 (Joanna Andrulonis); Tr. at 1527–28 (DeTommasi).

571. Tr. at 1579 (Rosen).

patient's attention for any period of time. His restlessness and agitation between hospitalizations made it difficult to engage Andrulonis in any therapeutic tasks.[572] Following his discharge after his second hospitalization, Andrulonis has remained agitated and confused.[573] After more than ten years of therapy, the pathologist is now able to hold Andrulonis' attention for up to one-half hour in weekly sessions.[574] Andrulonis, who has the vocabulary of a three to four year old,[575] is able to name letters, identify some pictures, and speak words reproduced on a printed page.[576] There is little or no evidence that Andrulonis has any comprehension of the words he repeats.[577] Andrulonis engages in "overlearned" or "automatic" speech, repeating words or phrases that he had become accustomed to using without thinking before the onset of his illness.[578] Thus, Andrulonis will greet people by saying "hello."

572. Tr. at 1581-82 (Rosen).

573. Tr. at 1584 (Rosen).

574. Tr. at 1585, 1591-92 (Rosen).

575. Tr. at 1597 (Rosen).

576. Tr. at 1587, 1589-90 (Rosen).

577. Tr. at 1590 (Rosen).

578. Tr. at 1586 (Rosen).

Swearing is commonly an aspect of automatic speech, and Andrulonis swears frequently. Andrulonis, whose two daughters were around the ages of nine and six at the time he first became ill, will repeat the phrase, "You are the cutest little girl in the world," but the sentence will be stripped of its meaning by its use in inappropriate circumstances.[579]

The anatomical brain damage Andrulonis suffered as a result of rabies encephalitis is permanent. The nervous tissue Andrulonis has lost will not regenerate itself. Given the profound loss of brain matter he has suffered and his failure to exhibit any significant improvement in intellectual function in the more than ten years following his illness, it is unlikely that that part of Andrulonis' brain that was not damaged as a result of his illness will compensate for the loss of function accompanying his cortical atrophy.[580] Consequently, the court must conclude that the loss of higher intellectual function that has attended the anatomical damage Andrulonis suffered is also permanent.[581] Andrulonis has suffered virtually a complete memory loss regarding

579. Tr. at 1588 (Rosen).

580. Tr. at 849–50 (Rodichok); Tr. at 730 (Tillotson).

581. Tr. at 859–60 (Rodichok); Tr. at 729–30 (Tillotson).

events that preceded his initial hospitalization in April 1977, and that memory loss is permanent.[582] The personality and behavioral changes that resulted from the disease Andrulonis acquired are also permanent.[583] Andrulonis suffers from dementia secondary to rabies encephalitis, and the dramatic emotional and behavioral instability associated with his dementia that has been described above will persist for the remainder of his life.[584]

Andrulonis continues to exhibit impatience and has a poor attention span; his short-term memory is poor; his judgment is impaired, and he has no safety awareness whatsoever; his demeanor is usually either detached and uncommunicative or agitated and restless.[585] Andrulonis continues to have violent outbursts which are usually directed at his wife, although the frequency of those outbursts have decreased significantly.[586] Andrulonis now

582. Tr. at 860–61 (Rodichok).

583. Tr. at 1531 (DeTommasi); Tr. at 860 (Rodichok).

584. Tr. 1529–31 (DeTommasi).

585. Tr. at 1541, 1545–46 (DeTommasi); Tr. at 1594–95 (Rosen).

586. Tr. at 1544–45, 1546–47 (DeTommasi); Tr. 1764–65 (Joanna Andrulonis).

shows remorse after pushing or striking his wife.[587] He continues to experience dramatic mood swings; he still engages in repetitive movements, paces, and constantly rocks back and forth while seated.[588]

There is no evidence that Andrulonis has spontaneously displayed any significant goal-directed activity at any time since he contracted the disease of rabies; rather, he has consistently engaged in nondirected behavior such as staring at his feet or pulling on his socks.[589] He does not function well absent a predictable structure provided by his home, family and others with whom he is familiar. He lacks the cognitive ability to initiate most activities, including self-care activities. He cannot shower himself without assistance; he can help dress himself if someone hands him an article of clothing and instructs him to put it on; he cannot prepare his own meals, and must still be monitored while he eats; and he is unable to make use of the toilet without help.[590] Andrulonis now requires adult su-

587. Tr. at 1745 (Joanna Andrulonis).

588. Exhs. 158, 159 ("Day-in-the-Life" Videotape).

589. *See* Tr. at 1526, 1541 (DeTommasi); Tr. at 1595 (Rosen); *see also* Exh. 158 (Day-in-the-Life Film).

590. Tr. at 1748–49 (Joanna Andrulonis); Tr. at 1595 (Rosen); Exhs. 158, 159.

pervision at all times, and will require such supervision for the rest of his life.[591]

Currently, Andrulonis receives therapy from a speech language pathologist once a week for one-half hour. Andrulonis apparently cannot presently tolerate more than one-half hour of such therapy at a time.[592] Since the onset of his illness, Andrulonis has had difficulty pronouncing words and has exhibited a comparative inability to recognize objects or use words appropriately.[593] Andrulonis can name all of the letters of the alphabet and identify the numbers one through ten, and can identify colors when engaging in certain types of therapy but not in others. Tests consistently show that he has the vocabulary ordinarily possessed by a three or four-year old infant, but that he is unable to express ideas or communicate desires or discomforts to others.[594]

Overall, the progress Andrulonis has made as a result of his speech therapy has been limited. Nonetheless, continued therapy will likely benefit him. Certain of An-

591. Tr. at 1529–30 (DeTommasi); *see also* Tr. at 1376 (Piedmont); Tr. at 1760 (Joanna Andrulonis).

592. Tr. at 1577 (Rosen).

593. Tr. at 1532 (DeTommasi).

594. Tr. at 1597–98 (Rosen).

drulonis' specific behaviors will likely improve if such therapy is continued. He has gradually extended his attention span over the past decade to the point where he can occasionally be engaged in a task for twenty to thirty minutes, and such incremental improvement can be expected in the future. Certain simple pre-writing skills such as word recognition may also improve in the future.[595] The long-term prospect for improvement, however, is necessarily limited by the extent of the anatomical damage to the brain Andrulonis has suffered.[596]

At the time of trial, Andrulonis received physical and occupational therapy at AMCH designed to improve his motor skills and his ability to imitate directions given by others, and the progress made in this area has apparently been steady.[597] Andrulonis continues to take a regimen of drugs designed to control his behavior and calm his agitation, and he will require such drugs for the rest of his life.[598] A possible future side-effect of the persistent use of the anti-psychotic drugs administered to

595. Tr. at 1594–97; *see also* Tr. at 1532 (DeTommasi).

596. Tr. at 730 (Tillotson).

597. Tr. at 1756–57 (Joanna Andrulonis); Tr. at 1595 (Rosen).

598. Tr. at 1531 (DeTommasi).

Andrulonis is the development of an uncomfortable and disabling neurologic impairment known as tardive dyskinesia.[599] Andrulonis currently is treated by a neurologist who monitors his medication and treats his emotional problems. The neurologist sees Andrulonis an average of eight to nine times a year, and this level of treatment will be necessary for the remainder of Andrulonis' life.[600]

B. *The Calculation of Damages*

[73] Damages in FTCA actions "are determined by the law of the State where the tortious act was committed, 28 U.S.C. § 1346(b), subject to the limitations that the United States shall not be liable for 'interest prior to judgment or for punitive damages,' 28 U.S.C. § 2674." *Hatahley v. United States*, 351 U.S. 173, 182, 76 S.Ct. 745, 752, 100 L.Ed. 1065 (1956); *see also Ulrich v. Veterans Administration Hospital*, 853 F.2d 1078, 1081–82 (2d Cir.1988). Under New York law, a plaintiff who suffers personal injury as the result of the actionable negligence of another may recover damages for the loss of earning capacity, medical expenses incurred and conscious mental and physical pain and suffering. *Ulrich*, 853 F.2d at 1082 (citing 36 N.Y.Jur.2d *Damages* § 57, at 102–03 (1984)). The court will assess each of these elements of damages in turn.

599. Tr. at 155 (DeTommasi).

600. Tr. at 1525, 1531–32 (DeTommasi).

1. Loss of Earning Capacity

[74] For the most part, the court finds the expert testimony regarding economic damages offered by plaintiffs' economist, Dr. Thomas R. Kershner, more convincing than that of the Government's economist, Dr. Paul C. Grier. The court does not, however, accept all of the assumptions made by plaintiffs' expert in rendering his opinion and finds that aspects of the opinion testimony offered by Dr. Grier are well-considered. Consequently, the court will discuss in some detail those assumptions it finds are supported by the record.

It is uncontested that Andrulonis is permanently and totally disabled, has not been employed since April 1977 and will never work again. The first conflict between the two experts that must be resolved concerns the work life expectancy of Andrulonis. The Government's expert assumed that Andrulonis would be gainfully employed until he reached 62.6 years of age. This assumption was based on a work life expectancy table published by the Department of Labor in 1976.[601] Reliance on a report prepared in 1976 is fatally flawed, however, since at that time employers were allowed by federal law to impose mandatory retire-

601. Tr. at 1897 (Testimony of Dr. Paul C. Grier).

ment at age 65, whereas today most employers—including NYSDOH—are, with certain exceptions, precluded from imposing such arbitrary age limits on an employee's work life.[602] Dr. Kershner assumed Andrulonis would work until age 65, but this assumption also was not supported with adequate data.[603] Absent a reliable statistical basis for either opinion, the court assumes that Andrulonis would have worked until age 65 if not for his tragic accident. This judgment is based in part on the court's sense (admittedly neither supported nor controverted by available data) that the statutory prohibition of a mandatory retirement age will increase the average work life of most workers. Further, Andrulonis was engaged in a line of employment in which one could likely pursue a career late into life and which ordinarily is not considered unusually hazardous. For convenience, the court will assume that had he not suffered his injuries Andrulonis would have worked through the year 2007.[604]

602. *See* Age Discrimination in Employment Act, 29 U.S.C. § 621 *et seq.*; *id.* §§ 630(b), 631(a); Tr. at 1917 (Grier); Tr. at 1615–17 (Testimony of Dr. Thomas R. Kershner).

603. *See* Tr. 1615–17 (Kershner).

604. If Andrulonis survives, he will turn 65 on February 27, 2008.

[75, 76] To determine appropriate compensation for the lost earnings Andrulonis has suffered and will suffer in the future, the court must first estimate "what the lost stream of income would have been" from the time Andrulonis was initially hospitalized until the end of the year 2007. *See Jones & Laughlin Steel Corp. v. Pfeifer*, 462 U.S. 523, 536, 103 S.Ct. 2541, 2550, 76 L.Ed.2d 768 (1983) (a non-FTCA case applying general principles). For the calculation of future lost earnings, the "lost stream of income" is to be determined by assuming an inflation-free economy; the effect of inflationary forces in calculating a lump sum damage award is considered in arriving at a discount rate to be used in reducing the award for future lost earnings to present value. *Id.* at 536–38, 103 S.Ct. at 2550–51. Courts generally approximate the stream of income as a series of annual after-tax payments. *Id.* at 536, 103 S.Ct. at 2550. Each annual "installment" is comprised of the injured party's projected wage for that year had he not been disabled as well as fringe benefits such as insurance coverage, pension and retirement plans, and health insurance. *Id.* at 534 n. 12, 103 S.Ct. at 2549 n. 12; *see Dennis v. Dachs*, 85 A.D.2d 223, 448 N.Y.S.2d 1 (1st Dept.1982) (testimony regarding lost wages and fringe benefits allowed). Each annual installment is to be diminished by the state and federal taxes the injured party could

be expected to pay.[605] *Pfeifer*, 462 U.S. at 534, 103 S.Ct. at 2549.

605. Under New York law, the lost wages component of a damage award in a personal injury or wrongful death action is to be calculated on the basis of gross projected earnings, with no reduction for likely future taxes. *Johnson v. Manhattan & Bronx Surface Transit Operating Authority*, 71 N.Y.2d 198, 206, 524 N.Y.S.2d 415, 419, 519 N.E.2d 326, 330 (1988); *see also Woodling v. Garrett Corp.*, 813 F.2d 543, 557–58 (2d Cir.1987) (applying New York law). Ordinarily, the FTCA requires a court to follow state law in determining damages, but a majority of courts have held that as a matter of federal law potential income tax consequences must be considered in making an award for lost compensation notwithstanding applicable state law concerning that issue. *See, e.g., Shaw v. United States*, 741 F.2d 1202, 1206 (9th Cir.1984); *Flannery v. United States*, 718 F.2d 108, 111 (4th Cir.1983), *cert. denied*, 467 U.S. 1226, 104 S.Ct. 2679, 81 L.Ed.2d 874 (1984); *O'Connor v. United States*, 269 F.2d 578, 584–85 (2d Cir.1959); *Barrett v. United States*, 660 F.Supp. 1291, 1318 (S.D.N.Y.1987). These cases reason that because a damage award in a personal injury suit is not considered taxable income, *see* 26 U.S.C. § 104(a)(2), the failure to deduct income taxes in an FTCA award would "result in the imposition of punitive damages against the government" in contravention of 28 U.S.C. § 2674. *Shaw*, 741 F.2d at 1206. This proposition is not free from doubt. One court has argued that the decision whether to deduct income taxes in determining fair compensation for an injury is but one of many issues affecting the amount of the ultimate award that are completely unrelated to the traditional understanding of "punitive damages" as that term is

[77] With regard to the calculation of Andrulonis' net inflation-free lost earnings, a second conflict arises between the economists who testified in this case. Dr. Grier assumed that Andrulonis would remain employed by NYSDOH for the rest of his work life and that he would not leave the Bacteriologist series. The economist assumed that Andrulonis would remain at grade 18 until 1980, that in 1980 he would be promoted to grade 23, and that he would remain at grade 23 for the rest of his

used in the law, and that through § 2674 Congress forbade "punitive damages" only as the concept is understood "in its traditional sense." *Manko v. United States*, 830 F.2d 831, 836 (8th Cir.1987); *see also Flannery*, 718 F.2d at 113–15 (Hall, J., dissenting); *Rufino v. United States*, 829 F.2d 354, 362 (2d Cir.1987) ("[T]he FTCA's prohibition of punitive damages was designed to prohibit 'use of a retributive theory of punishment against the government.'" (quotation omitted)). Nonetheless, perhaps perceiving themselves bound by the Second Circuit's decision in *O'Connor*, the parties in this case offered expert opinion testimony based on net, or after-tax, income estimates.

career.[606] Dr. Kershner, on the other hand, calculated annual installments on the basis of a 1985 study conducted by a private research group on behalf of the Department of Energy. The study determined the nationwide average salaries for scientists and engineers in various fields who worked in the United States. Plaintiffs' economist was able to discern from this study the average salary of biologists with a master's degree at different stages in their careers.[607]

The court accepts the assumptions of plaintiffs' expert on this issue. It is clear that the Government's economist had not been made aware of the options for advancement open to Andrulonis had he stayed with NYSDOH for the entirety of his career.[608] Biologists with master's degrees employed by the state are not locked into the Bacteriologist Series; indeed, the court is aware, for example, that Andrulonis possessed the minimum educational re-

606. Tr. at 1901–03, 1905–06, 1928–31 (Grier).

607. Tr. at 1608–09, 1636, 1647–48 (Kershner).

608. *See* Tr. at 1928–31 (Grier); *see also* Tr. at 1645–46 (Kershner).

quirements to move into the Research Scientist Series to which Dr. Debbie belonged, although he had not taken or passed the appropriate civil service examination given for those positions.[609] Early in his career, Andrulonis had shown notable ambition. He completed the studies for his master's degree while working full time at Griffin at a time when he and his wife were starting a family. There is no reason to believe Andrulonis would lose that ambition and remain in a lower paying job series indefinitely. Moreover, to accept the scenario presented by the Government, one would have to assume that Andrulonis would remain at NYSDOH at the lower grades the Government's economist assumed while resisting the economic incentives of greater pay in the private sector or in another geographical area. While the record indicates that Andrulonis was happy living in the Albany area and enjoyed his work at Griffin, there is no indication that his ties to either that geographical region or NYSDOH were so strong that he would make the economic sacrifices the Government would like the court to assume.

609. *See* Exh. 1 (New York State Department of Civil Service, "Classification Standard for Research Scientists" (revised August 1981)).

The assumptions made by plaintiffs' economist concerning Andrulonis' lost stream of income are preferable in one other particular. The Supreme Court has noted that

> [e]ven in an inflation-free economy—that is to say one in which the prices of consumer goods remain stable—a worker's wages tend to "inflate." This "real" wage inflation reflects a number of factors, some linked to the specific individual and some linked to broader societal forces. With the passage of time, an individual worker often becomes more valuable to his employer. His personal work experiences increase his hourly contributions to firm profits. To reflect [this], he will often receive "seniority" or "experience" raises, "merit" raises, or even promotions....

Pfeifer, 462 U.S. at 535, 103 S.Ct. at 2549 (footnotes omitted); *see also Dennis*, 85 A.D.2d 223, 448 N.Y.S.2d 1 (specific evidence of wage increments permissible under New York law). The testimony concerning Andrulonis' work habits and the high regard in which he was held by his superiors before he contracted his illness is strong evidence that Andrulonis would likely have benefited from such "real" wage increases over time. Consequently, the comparatively static wage assumptions made by Dr. Grier seem inadequate. The wage assumptions made by Dr. Kershner better reflect Andrulonis' potential to receive annual salaries that would reflect the

increasing value a progressively more experienced employee has to an employer.

[78] With regard to the estimation of the value of the fringe benefits Andrulonis would have received had he not become totally disabled, the economists again disagreed. Dr. Kershner assumed that the value of Andrulonis' fringe benefits each year would approximate thirty percent of the net salary he earned that year.[610] Dr. Grier assumed that fringes should be valued at 26.66 percent of Andrulonis' after-tax salary.[611] The court finds Dr. Grier's testimony on this point unconvincing. Grier conceded that for the average employee working for the State of New York—the economist had assumed Andrulonis would work for the State for his entire career—the value of fringe benefits approximated thirty percent of net salary.[612] Grier offered no convincing reason for deviating from this average. He argued that as an individual's salary increases, the percentage relationship of fringe benefits to wages decrease because the cost of certain

610. Tr. at 1648–51 (Kershner).

611. Tr. at 1900–01 (Grier).

612. Tr. at 1936 (Grier). The State's objection to this testimony is overruled.

benefits such as health insurance [613] remain constant regardless of the salary of the individual receiving those benefits.[614] There is nothing in the record indicating when, if ever, in the salary projections for Andrulonis that Grier submitted to the court that the ratio of wages to fringe benefits would fall below 10:3. In light of Dr. Kershner's testimony that an even higher ratio between wages and fringes than 10:3 could have been assumed, the court will accept the assumption Kershner did make in his opinion testimony.[615]

613. Both experts included health benefits in their evaluation of Andrulonis' lost fringe benefits. Although the record is unclear on the point, the court assumes that these benefits would cover the cost of medical services for the Andrulonis family that are unrelated to Andrulonis' rabies encephalitis, since the latter costs are incorporated in the court's award for medical expenses, *see* text, *infra* at 1521–23, and since both economists rendered opinions about the cost of the medical expenses attributable to the illness Andrulonis has suffered as well as the cost of an identical benefit package that included health insurance. *See* Tr. at 1900 (Grier) (experts assumed the same benefit package).

614. Tr. at 1901–02, 1937 (Grier).

615. *See* Tr. at 1648–51 (Kershner).

[79–81] Once the stream of annual after-tax income in a hypothetical inflation-free economy has been determined, the court must discount each annual installment estimated for future years to present value and add the installments together to determine the total lump sum award for future lost earnings.[616] *See Pfeifer*, 462

616. The Government presented the testimony of an agent licensed by the State of New York to sell life insurance, annuities, and the like, and this agent offered evidence concerning the cost of purchasing an annuity from a private insurance company that would "guarantee" a stream of payments that would continue for life. Tr. at 1989–2011 (Testimony of Paul A. Hoffman). This testimony, offered as an "alternative" to the application of a discount rate, is not helpful for a number of reasons.

Initially, it should be noted that the FTCA contemplates a lump-sum award, and this court lacks the power to enter judgment in this case "in terms other than a simple award of money damages," and thus cannot mandate the purchase of an annuity or otherwise structure its award. *Frankel v. Heym*, 466 F.2d 1226, 1228 (3d Cir.1972); *Reilly v. United States*, 665 F.Supp. 976, 1017 (D.R.I.1987), *modified on other grounds*, 863 F.2d 149 (1st Cir.1988); *see also Pfeifer*, 462 U.S. at 533, 103 S.Ct. at 2548 (In action brought under § 4 of the Longshoreman's and Harbor Worker's Compensation Act, 33 U.S.C. § 904, Supreme Court noted that "[t]he award could in theory take the form of periodic payments, but in this country it has traditionally taken the form of a lump sum, paid at the conclusion of the litigation."). While potential investment options are considered by the court in calculating a lump-sum amount approximating economic loss when it

U.S. at 537–38, 103 S.Ct. at 2550–51; *Dullard v. Berkeley Associates Co.*, 606 F.2d 890, 895 n. 4 (2d Cir.1979). The Second Circuit has indicated that in FTCA cases New York law "governs the issue of dis-

determines the appropriate discount rate to apply, conservative investment strategies are presumed. *See Pfeifer*, 462 U.S. at 537, 103 S.Ct. at 2550–51. The court is unaware of any case in which the purchase of long-term annuities from private companies that are not guaranteed by the Government was deemed the sort of "conservative" investment option contemplated by this rule, either in terms of the risk of default or the risk that future inflation would be higher than anticipated at the time the annuity was purchased. *But see* N.Y.C.P.L.R. §§ 5031, 5041 (McKinney Supp.1989).

In any event, the agent who testified about the cost of the proposed annuity lacked knowledge of most of the underlying assumptions upon which the figures he offered were based, did not consider the tax consequences associated with the payments made from the annuity, and indicated that the calculation on the annuity was based on the unexplained assumption that Jerome Andrulonis was a "substandard risk" (would likely have a reduced life expectancy), even though the evidence does not support this assumption. The agent also assumed a flat growth rate in Andrulonis' salary of five percent per year, which arguably adjusts for inflation but ignores increases in salary which could have been expected on the basis of experience and future promotions. Tr. at 1990–91 (Hoffman). On the whole, these shortcomings leave the court unpersuaded by the agent's testimony, and the court will disregard it in assessing damages in this case.

counting," *O'Rourke v. Eastern Airlines, Inc.*, 730 F.2d 842, 857 n. 24 (2d Cir.1984), and while evidence of the present value of lost future earnings must be considered under the law of New York, *see Hanratty v. City of New York*, 132 A.D.2d 596, 517 N.Y.S.2d 757 (2d Dept.1987), there is little case law from New York state courts concerning the method of determining a discount rate. *See Dennis*, 85 A.D.2d at 225–27, 448 N.Y.S.2d at 2–3. Other courts applying New York law, however, have indicated that the discount rate should not only account for the income the plaintiff will earn in the future from the investment of a lump sum award but also the effect future inflation will have on the purchasing power of money.[617] *See, e.g., Woodling v. Garrett Corp.*, 813 F.2d 543, 559 (2d Cir.1987); *O'Rourke*, 730 F.2d at 857.

[82] For the calculation of lost future earnings, Dr. Grier assumed a discount rate of one percent, while Dr. Kershner assumed a discount rate of negative .18 percent (effectively increasing rather than

617. The refusal of the New York courts to allow the introduction of evidence concerning the effects of taxation on future lost earnings because such evidence is considered "speculative," however, might lead state courts to disregard potential inflationary forces for the same reason. *See supra* note 605.

decreasing the future after-tax stream of income estimated for Andrulonis).[618] The Second Circuit has held that two percent was the lowest discount rate it was willing to accept "in the absence of historical data justifying a different rate." *Doca v. Marine Mercante Nicaraguense, S.A.*, 634 F.2d 30, 40 (2d Cir.1980), *cert. denied*, 451 U.S. 971, 101 S.Ct. 2049, 68 L.Ed.2d 351 (1981). In contrast to his testimony concerning the proper discount rate to be applied to future medical expenses,[619] Dr. Kershner did not provide the court with an adequate basis for his testimony regarding the proper discount rate to be applied to an award for lost future earnings. Consequently, the court will apply the two percent discount rate suggested by the Second Circuit in *Doca* to Dr. Kershner's projections concerning Andrulonis' future wages and fringe benefits.[620] The court's method

618. *See* Tr. at 1898 (Grier).

619. *See* text, *infra* at 1522.

620. The court has not adopted the one percent discount rate suggested by Dr. Grier because it is not clear to the court whether that rate takes into account a projected increase in medical costs. Because the court has adopted the rate for calculating medical costs advocated by Dr. Kershner, *see* text, *infra* at 1522, to give plaintiffs the benefit of Dr. Grier's assumption could potentially result in "double-counting" the effect of spiralling medical costs on the inflation rate as a whole.

of calculation is discussed in the margin.[621]

621. Dr. Kershner's work sheets were not admitted into evidence. His opinion concerning future lost wages was offered as a lump sum after application of the discount rate that the court has not accepted. Thus, the court is not aware of the specific assumptions Kershner made concerning Andrulonis' projected annual salary for each year between 1987 and 2007. The court does know, however, that Kershner assumed an after-tax salary of $13,659 for 1978 and an after-tax salary of $17,346 for 1979. Tr. at 1636 (Kershner). The court also knows that the gross salary projected for the year 2007 (assuming an inflation-free economy) was $67,176, and that Kershner deducted 19.7 percent from each annual installment for state and federal taxes. Tr. at 1620, 1648 (Kershner). This would result in a net salary of $53,942 projected for the year 2007. If it is assumed that Andrulonis' inflation-free salary rose at a uniform rate from $17,346 in 1979 to $53,942 in 2007, Andrulonis' "real" wage inflation would be $1,307 a year. Assuming this uniform rise, Andrulonis would have received an after-tax salary of $18,653 in 1980, $19,960 in 1981, $21,267 in 1982, and so forth. The projected after-tax salary for 1987 would be $27,802, and 1987 will be the first year to which the two percent discount rate will be applied, since the trial in this case was conducted that year and the assumptions of the economists were couched in terms of present value in 1987.

To reduce each annual installment to present (1987) value, the projected installment is multiplied by the discount factor of $1/(1 \pm i)^n$, where i = the discount rate (two percent, or .02), and n = the year for which present value is sought. After the discount factor is applied to each annual installment, the discounted installments are added together. *See Dullard v. Berkeley Associates Co.*, 606 F.2d 890, 895 n. 4 (2d Cir. 1979).

On the basis of the foregoing, the court accepts Dr. Kershner's opinion that the value of Andrulonis' net lost wages through 1986 is $206,638. The court finds that the present value of future net wages Andrulonis has lost as a result of his disability, as measured from the time of the trial, is $679,164. The court finds that the present value of the lost fringe benefits Andrulonis has suffered and will suffer is $265,741.

2. Medical Expenses

The value of the services provided Jerome Andrulonis by AMCH in connection with his initial hospitalization for rabies encephalitis and his subsequent admissions for complications resulting from this condition is approximately $87,000.[622] The value of the medical services rendered Andrulonis by his treating physician during his initial hospitalization is $4,400.[623] An additional $406,454.89 was expended for Andrulonis' medical care and support costs through February, 1987.[624] Thus, the past medical expenses Andrulonis incurred as a result of his illness total $497,854.89.

622. Tr. at 809 (Tillotson).

623. Tr. at 731 (Tillotson).

624. Tr. at 1566 (Testimony of Betsy G. Crawford); Exh. 122 (Summary of medical & claim adjustment expense payments made on behalf of claimant Jerome Andrulonis by New York State Insurance Fund).

[83] In order to estimate the future medical expenses Andrulonis will incur as a result of his illness, the court must make an assumption regarding his life expectancy. The evidence regarding Andrulonis' projected life span is not consistent. The physician who treated Andrulonis during his first hospitalization at AMCH testified that Andrulonis was more vulnerable to acquiring pneumonia, more apt to fall and injure himself, and more likely to be exposed to the dangers associated with hospitalization than the average person, and thus a shortened life span could be expected.[625] On the other hand, two neurologists who have treated Andrulonis testified that Andrulonis' physical and mental condition was not likely to affect his life expectancy.[626] In fact, one of the neurologists indicated that Andrulonis, if properly supervised, would have a reduced risk of accidental death, thus implying that his life expectancy might be somewhat greater than the average person.[627] The neurologist who

625. Tr. at 745–46 (Tillotson).

626. Tr. at 1533 (DeTommasi); Tr. at 868–69 (Rodichok).

627. *See* Tr. at 869–70 (Rodichok).

was treating Andrulonis at the time of trial testified that except for victims of Alzheimer's disease, there is no evidence that individuals who have suffered a brain injury of the sort Andrulonis has endured, whether caused by trauma or infection, will have either an increased or decreased life span.[628] On the whole, the evidence concerning life expectancy is inconclusive, and thus the court will assume a normal life span in calculating damages for projected medical costs and future pain and suffering. At the time of trial, actuarial tables indicated that the remaining life expectancy of Jerome Andrulonis was 30.9 years.[629]

Dr. Kershner estimated that assuming a normal life span Andrulonis will incur future medical expenses totalling $167,812 after being adjusted to present value.[630] This total covers the cost of consultations with physicians as well as the cost of various drugs and medications. In arriving at this total, Kershner projected the amount expended on such costs in 1986 through the year 2017 and applied a negative discount rate of 1.88 percent.[631] Kershner derived this rate by comparing the rates of return on short-term municipal bonds with the in-

628. Tr. at 1559–61 (DeTommasi).

629. Tr. at 1769.

630. Tr. at 1616 (Kershner).

631. *See* Tr. at 1663–65, 1612 (Kershner).

creases in medical services prices. Kershner studied the relationship of these two rates over a thirty year period. He found that the rate of increase in the costs of medical services—which has been markedly higher than inflation in general—surpassed the interest obtained from short-term bonds by an average of 1.88 percent, and he projected that this thirty year trend would continue in the future.[632] The court finds this prediction to be sound, and accepts Kershner's testimony regarding future medical expenses.

Dr. Kershner estimated that the present value of the physical, occupational, and speech therapy Andrulonis should receive is $288.232, as calculated by applying the negative 1.88 percent discount rate.[633] Kershner was asked to assume that Andrulonis will require one hour of speech therapy a week in the future, even though he presently can tolerate only one-half hour of such therapy a week.[634] Although Andrulonis' attention span has improved, albeit at a painstakingly slow pace, and although he might tolerate an hour's speech therapy a week in the future, this supposition was not supported by expert medical testimony

632. Tr. at 1612–13, 1623–24 (Kershner).

633. Tr. at 1613–14 (Kershner).

634. Tr. at 1661 (Kershner).

adduced at trial. Accordingly, the court will reduce Kershner's projection on this element of future damages by one-sixth, approximating the proportion of the estimated cost of therapy attributable to the extra one-half hour of speech therapy the economist assumed.[635] The court otherwise accepts Kershner's assumptions concerning the valuation of the costs of future therapy.

There is also no expert medical testimony supporting the contention that Andrulonis requires care by three shifts of licensed practical nurses (LPNs). While Andrulonis has in the past required the care of LPNs at least one quarter of the time, plaintiffs have not offered competent expert testimony establishing the continued need for such care. Joanna Andrulonis, in fact, testified that Andrulonis does not necessarily require aides with a nursing background.[636]

[84, 85] The Government's suggestion that Andrulonis requires no more than one shift of a home health aide to care for him, however, is simply preposterous. It cannot be seriously disputed that Andrulonis requires constant around-the-clock care and supervision by an adult. The Government invites the court to impose on Joanna An-

635. *See* Tr. at 1662 (Kershner).

636. Tr. at 1759–60 (Joanna Andrulonis).

drulonis the legal obligation to provide that care sixteen hours a day for the rest of her life. The court declines the invitation. Joanna is under no legal obligation to provide such services. That she has sacrificed so much to care for her husband through the years since his illness speaks volumes of her strength and devotion, but this dedication does not evidence an assumption of a duty to relieve the Government or the State of responsibility for the damage done by the negligence of their employees. Indeed, as her children leave home and as she gets older, her ability to provide the care she has provided in the past will likely decline. Jerome Andrulonis is entitled to three eight-hour shifts of aides to provide him the custodial care he requires.

Determining the cost of such care has been made problematical by Joanna Andrulonis' inability to retain dependable workers who could be entrusted with the responsibility of caring for her husband. For several years the New York State Insurance Fund has been willing to reimburse Joanna Andrulonis for the cost of three shifts of home health aides.[637] For most of that time, Joanna Andrulonis has only been able to find suitable aides for one or two shifts a day, and the aide who currently cares for her husband is an LPN who is paid the salary of an LPN.[638] Joanna testi-

637. *See* Tr. at 1746 (Joanna Andrulonis).

638. Tr. at 1757 (Joanna Andrulonis).

fied that it was difficult to find aides who could be depended upon to show up for work, that a frequent turn-over of aides disrupted the predictable and stable environment her husband requires, and that some of the aides hired were of questionable character.[639] Indeed, one such aide stole money and jewelry from the Andrulonis family over a period of six months, and another came to work intoxicated and made sexual advances toward one of the Andrulonis children.[640]

[86] The fact that Joanna Andrulonis has been unable to fill the home health aide positions for most of the past decade even though the State Insurance Fund had agreed to pay for them indicates that in the Albany-area market there is a lack of individuals available at the wages paid home health aides who are qualified to provide the custodial care to which Jerome Andrulonis is entitled. The evidence also indicates that Joanna was able to maintain three shifts of care-providers who furnished the stable and consistent environment Andrulonis prefers when the State Insurance Fund was willing to pay for LPNs.[641] The court must conclude that the only individuals Joanna Andrulonis has

639. Tr. at 1747–48, 1757–59 (Joanna Andrulonis).

640. Tr. at 1758–59 (Joanna Andrulonis).

641. *See* Tr. at 1738 (Joanna Andrulonis).

been able to find who can adequately care for her husband earn salaries approximating that of LPNs working in the Albany area, and the court's estimate of the cost of future custodial care will be based on the assumption that the services of dependable home health aides cannot be obtained at less than the salaries earned by LPN's in the Albany area.

In 1987, LPNs earned $11.75 per hour in the Albany area.[642] Providing three eight-hour shifts of custodial workers for each day of the rest of Andrulonis' projected life span would cost $103,000 each year until the year 2018, assuming an inflation-free economy. Because plaintiffs have failed to establish that the custodial aides caring for Andrulonis must have a nursing background, it is inappropriate to apply the discount rate Dr. Kershner calculated for future medical services (negative 1.88 percent). The court will instead apply the two percent discount rate it used in determining the present value of Andrulonis' future lost earnings. The court's calculations indicate that the present value of the custodial care to which Andrulonis is entitled is $2,417,238.

[87, 88] The court will not reduce plaintiff's recovery for economic losses by the amount of the payments he and his children have received as Social Security bene-

642. Tr. at 1656 (Kershner).

fits. Whether such benefits are deducted from an award under the FTCA depends on the applicable "collateral source" rule of the state whose law governs. *Douglas v. United States*, 658 F.2d 445, 449 n. 5 (6th Cir.1981); *Smith v. United States*, 587 F.2d 1013, 1016 (3d Cir.1978); *Klein v. United States*, 339 F.2d 512, 517–18 (2d Cir.1964). In actions commenced prior to June 28, 1986, New York law provided that damages recovered by an injured party from a tortfeasor would not be reduced by non-gratuitous compensation that is received from a third party for losses associated with the injury. *Healy v. Rennert*, 9 N.Y.2d 202, 206–08, 213 N.Y.S.2d 44, 46–48, 173 N.E.2d 777, 778–80 (1961); *Castleberry v. Hudson Valley Asphalt Corp.*, 60 A.D.2d 878, 879, 401 N.Y.S.2d 278, 279 (2d Dept.1978); *Silinsky v. State-Wide Ins. Co.*, 30 A.D.2d 1, 4, 289 N.Y.S.2d 541, 546 (2d Dept.1968); *Anastasia v. Barnes*, 127 Misc.2d 971, 973–74, 487 N.Y.S.2d 628, 630–31 (Sup.Ct.1985). The rationale for this rule is that a wrongdoer should not benefit because the injured party receives benefits from funds to which the injured party had contributed and the wrongdoer had not. *Anastasia*, 127 Misc.2d at 972, 487 N.Y.S.2d at 630; Restatement (Second) of Torts § 920A comment b. The cases have held that Social Security disability benefits and Medicare benefits are paid from a special fund to which employed persons contribute, and that that fund is separate and distinct

from general government revenues. Thus, such benefits are deemed payments from a collateral source. *See, e.g., Berg v. United States*, 806 F.2d 978, 984–86 (10th Cir. 1986); *Siverson v. United States*, 710 F.2d 557, 560 (9th Cir.1983); *Titchnell v. United States*, 681 F.2d 165, 176 (3d Cir.1982) (Medicare benefits); *Smith*, 587 F.2d at 1015–16 (Social Security benefits). The court rejects as frivolous the Government's argument that New York's abrogation of its collateral source rule for actions commenced on or after June 28, 1986, *see* N.Y. C.P.L.R. § 4545(c) (McKinney Supp.1989), somehow evidences that New York's prior rule "is effectively a penalty" that is barred by 28 U.S.C. § 2674, which prohibits awards against the Government for "punitive damages."

3. Pain and Suffering

Although the valuation of "tangible" damages such as lost earning capacity and medical expenses often cannot be accomplished without some measure of conjecture, it is the calculation of damages for conscious pain and suffering that is fraught with the greatest uncertainty. The distinctions drawn in cases involving the determination of this intangible element of compensatory damages often seem arbitrary, but this is inevitable when a theory of recovery is founded upon a pretense. As the New York Court of Appeals has observed, recovery for non-economic losses

such as pain and suffering

> is based on the legal fiction that money damages can compensate for a victim's injury. Although this device is as close as the law can come in its effort to right the wrong, it is still, nevertheless, a fiction for money will not replace or repair the lost or broken limb or remove the disability caused.

Howard v. Lecher, 42 N.Y.2d 109, 111, 397 N.Y.S.2d 363, 364, 366 N.E.2d 64, 65 (1977) (Wachtler, J.). When an individual's disability is as severe as that suffered by Jerome Andrulonis, this fiction seems particularly unsatisfactory. In cases involving catastrophic injuries, the award of damages for the intangible aspects of the injuries suffered is limited not by the belief that at some point the pain and suffering endured has been fully compensated but rather by the recognition that policy considerations ultimately require an upper boundary which a compensatory award cannot exceed.

[89, 90] Under the rubric of "pain and suffering," New York law permits recovery for a range of non-pecuniary losses including physical pain, the adverse emotional consequences attributable to that pain and the injury that caused it, and the frustration and anguish caused by the inability to participate in the normal pursuits and pleasures of life. *McDougald v. Garber*, 73 N.Y.2d 246, 253–57, 538 N.Y.S.2d 937, 939–41, 536 N.E.2d 372, 374–76 (1989); *see Gallo v. Supermarkets General Corp.*,

112 A.D.2d 345, 346–47, 491 N.Y.S.2d 796, 798 (2d Dept.1985). Recovery for this latter aspect of pain and suffering, referred to as the "loss of enjoyment of life," incorporates "the deprivation or impairment of the senses or of one's ability to engage in those activities and perform those functions which were part of the victim's life prior to the injury." *Rufino v. United States*, 829 F.2d 354, 359 n. 8 (2d Cir.1987) (quotation omitted). The New York Court of Appeals does not allow consideration of the loss of enjoyment of life resulting from an injury absent "some level" of cognitive awareness of the loss by the injured party. *McDougald*, 73 N.Y.2d at 255, 538 N.Y. S.2d at 940, 536 N.E.2d at 375. The court finds that this minimal level of perception is present in the case at bar. It can generally be said that Jerome Andrulonis has the cognitive ability possessed by an average infant between the ages of two and four, although his level of development varies depending on the particular skill or ability in question. He cannot communicate in words whether he appreciates to any degree the loss of his ability to participate in the normal pursuits and pleasures of his life before his illness, but the impatience, anger and frustration he frequently exhibits seems to be indicative of at least some level of awareness that he cannot do what he could do before his injuries. Consequently, the court will consider loss of enjoyment of life in attempting to measure Andrulonis' pain and suffering.

"Pain," as an element of non-pecuniary damages, has been defined as "the physiological response by the injured person to a corporal injury." *Rufino*, 829 F.2d at 359 n. 8 (quotation omitted). The distinguishing aspect of this case with regard to the pain Andrulonis suffered as a result of his illness was the period of extreme agitation —"raging," as it was described at trial[643]—that Andrulonis experienced on April 21, 1977, before he succumbed to coma. Rabies is a disease of the nervous system, and the unusual degree of agitation, thrashing, screaming and crying associated with the acute neurologic stage of the disease is perhaps the primary reason it has historically been one of man's most feared afflictions.[644]

Other than this period of apparent extreme pain, the physiological, non-emotional manifestations of Andrulonis' disease, while substantial, are not uncommon in cases involving serious injuries and long periods of hospitalization. For example, during Andrulonis' initial hospitalization and after he emerged from his coma, a second gastrostomy was performed; he suffered recurring urinary tract infection; he endured the frequent injection of needles and catheters that commonly accompa-

643. *See* Tr. at 1724 (Joanna Andrulonis).

644. *See* text, *supra* at 1437–38 and 1466.

nies hospitalization. In 1979, he underwent a cisternagram, a painful procedure involving the injection of radioactive material into the spinal fluid through a spinal tap.[645] During the periods of extreme agitation that followed his initial hospitalization, Andrulonis would frequently injure himself, developing bruises, shin splints, and cracked heels.[646] In the future, it is possible that he will endure an uncomfortable and permanent neurologic impairment that commonly accompanies the long-term administration of potent anti-psychotic drugs.[647]

The exceptional consequence of Andrulonis' illness, however, is not the purely physical pain and discomfort attributable to it but rather the extreme emotional disturbance Andrulonis has exhibited for the past decade and will likely experience for the remainder of his life. His often frenzied and desperate behavior has been recounted in some detail above.[648] Whether this agitated behavior is caused by fear, anxiety, pain, anger, frustration, or a combination of the same can only be surmised. This troubled, often violent behavior has been at

645. Tr. at 858–59 (Rodichok).

646. *See* text, *supra* at 1513.

647. *See* text, *supra* at 1516.

648. *See* text, *supra* at 1510–14.

times extreme and has persisted for twelve years. It evidences extraordinary emotional distress directly traceable to the severe brain injury Andrulonis has suffered.

It is also apparent that Andrulonis has experienced a marked loss of ability to participate in the normal pursuits and pleasures in which he engaged before his illness. It would seem, given his education and the nature of his chosen profession, that before he contracted the disease of rabies, Andrulonis was interested in intellectual endeavors; his ability to derive pleasure from intellectual activities has been virtually destroyed. While Andrulonis has regained much of his motor skills, safety concerns will limit his participation in the athletic activities he once enjoyed. He will no longer derive pleasure from the competitive aspects of athletics. He cannot fully appreciate the joys that he formerly experienced in connection with his family, church, and community activities.

[91] Assessing the entirety of Andrulonis' non-pecuniary loss, the court concludes that recovery from the pain and suffering associated with the disease of rabies and the anatomical damage caused by that disease should be at the upper end of the spectrum of reasonable awards for intangible injuries that have been rendered

in the State of New York.[649] The court

649. Some guidance can be drawn from other recent cases in New York involving non-pecuniary damage awards for catastrophic injuries. The plaintiff in *McDougald v. Garber*, 132 Misc.2d 457, 504 N.Y.S.2d 383 (Sup.Ct.1986), *aff'd*, 135 A.D.2d 80, 524 N.Y.S.2d 192 (1st Dept. 1988), *modified and remanded*, 73 N.Y.2d 246, 538 N.Y.S.2d 937, 536 N.E.2d 372 (1989), was a thirty-one-year-old woman who was deprived of sufficient oxygen while undergoing an elective caesarean section and as a result lapsed into a coma, from which she had not emerged at the time of trial. The plaintiff in that case is a permanent spastic quadriplegic. A jury award of $4,500,000 for conscious pain and suffering and loss of enjoyment of life was reduced to $2,000,000; the Court of Appeals remanded the case for a new trial on non-pecuniary damages because the trial court did not instruct the jury that some level of cognitive awareness is a prerequisite to recovery for the loss of enjoyment of life in New York. In *Gallo v. Supermarkets General Corp.*, 112 A.D.2d 345, 491 N.Y.S.2d 796 (2d Dept.1985), a jury awarded a young man in his twenties $1,400,000 for past and future pain and suffering resulting from injuries incurred when hot tar was spilled onto him at a jobsite. The victim suffered extensive third degree burns on his face, head, neck, chest, arms, and waist. He was hospitalized for nearly two months and subjected to various painful procedures such as debridement and skin grafting, and had three-quarters of his right ear removed. Permanent and extremely disfiguring facial scars resulted, as did severe psychological and mental problems. In a 1982 case, *Warmsley v. City of New York*, 89 A.D.2d 982, 454 N.Y.S.2d 144 (2d Dept. 1982), a forty-three-year-old woman who was involved in an automobile accident suffered a head injury, a chest injury, a fractured rib cage on both sides, a fracture of the sternum, a fracture of the clavicle, a broken left wrist and

finds that Andrulonis has experienced past pain and suffering for which an award of $1,200,000 is appropriate. The court determines that it is reasonably certain that Andrulonis will experience pain and suffering in the future, and finds that this pain and suffering should be compensated in the amount of $750,000. This figure is reached taking "the time value of money" into account, and the court here assumes that something approximating the two percent rate used in discounting future lost earnings is appropriate. *See Oliveri v. Delta S.S. Lines, Inc.*, 849 F.2d 742, 749-52 (2d Cir.1988).

forearm, and a severely fractured tibia and fibula in the right leg. She was initially hospitalized for forty days, underwent a tracheostomy, and endured various procedures to repair her seriously injured leg. Over the next three and one-half years, the plaintiff was hospitalized ten additional times for various procedures attempting to salvage her leg. She underwent eleven operations, the last of which resulting in the amputation of the right leg. The Appellate Division found that an award of $2,000,000 for past and future pain and suffering was warranted.

C. *Summary*

In sum, the court evaluates the damage suffered by Jerome Andrulonis as a result of the negligence of employees of the Government and the State as follows:

Past Lost Wages	$ 206,638
Future Lost Wages	679,164
Past and Future Lost Fringe Benefits	265,741
Past Medical Expenses (including therapy, nursing, and custodial care)	497,855
Future Medical Expenses	167,812
Future Physical, Occupational & Speech Therapy	$ 240,193
Future Custodial Care	2,417,238
Past Pain and Suffering	1,200,000
Future Pain and Suffering	750,000
Total	$6,42[illegible],641

[92, 93] The award to plaintiff Jerome Andrulonis will be satisfied by the United States. Because the non-governmental parties previously entered into settlement agreements with plaintiffs, the United States is entitled to have the total reward reduced in accordance with § 15–108 of New York's General Obligations Law. Under that provision, Andrulonis' damage award is reduced "to the extent of any amount stipulated by the release or the covenant, or in the amount of the consideration paid for it, or in the amount of the released tort-feasor's equitable share of the damages...." N.Y.Gen.Oblig.Law § 15–108 (McKinney 1978). No culpable

conduct attributable to either the WARF defendants or the Glatt defendants having been found, the court reduces Jerome's award by the amount in which his claims against those defendants were settled, $125,000. Defendants Lilly and Thompson & Sons[650] settled the claims of Jerome Andrulonis for a total of $100,000, which is less than the equitable share of the damages for which their actionable conduct was responsible. Thus, the court will reduce the award by 5%, the proportionate share of fault with which Lilly and Thompson & Sons has been charged, or a total of $321,232.

On the basis of the foregoing, plaintiff Jerome Andrulonis is awarded $5,978,409 on his claims against the United States. The United States is entitled to recover on its contribution claim an amount that does not exceed its equitable share of plaintiff's judgment, provided that third-party defendant NYSDOH is not required to pay more than its equitable share. N.Y.C.P.L.R. § 1402 (McKinney 1976). Therefore, the Government is entitled to recover $3,729,785 on its third-party claim against the State.

650. For purposes of calculating the off-set to which the Government is entitled, the court treats defendants Lilly and Thompson & Sons as a single entity. *Compare Mead v. Bloom*, 94 A.D.2d 423, 425-26, 464 N.Y.S.2d 904, 906 (4th Dept.1983), *aff'd*, 62 N.Y.2d 788, 477 N.Y.S.2d 326, 465 N.E.2d 1262 (1984).

VI. OTHER MATTERS

A. *The Government's Vacatur Motion*

Immediately before and during trial, plaintiffs entered into a series of three settlement agreements through which their claims against all of the non-governmental defendants were discontinued. In exchange, it was agreed that a total of $225,000 would be paid by the various non-governmental defendants in satisfaction of the primary claims of Jerome Andrulonis and $1,075,000 would be paid in satisfaction of the derivative claims of Joanna Andrulonis.[651] No objection to these settlements

651. On February 14, 1987, a settlement agreement between plaintiffs and the WARF defendants was reached. In exchange for terminating their claims against the WARF defendants, plaintiff Jerome Andrulonis was to receive $25,000 and plaintiff Joanna Andrulonis was to receive $175,000. On February 18, 1987, on the eve of trial, plaintiffs settled their claims against the Glatt defendants. Through this settlement agreement, plaintiffs discontinued their actions against the Glatt defendants, and in return defendant Glatt GmbH agreed to pay $100,000 in satisfaction of the claims of plaintiff Jerome Andrulonis and $400,000 in satisfaction of the derivative claims of plaintiff Joanna Andrulonis. On March 5, 1987, plaintiff settled their claims against defendant Eli Lilly and Company and defendant John L. Thompson and Sons and Company. Defendant Lilly agreed to pay $100,000 in satisfaction of the claims of plaintiff Jerome Andrulonis against both companies, and $500,000 to extinguish the claims of plaintiff Joanna Andrulonis against the two companies.

was voiced by either the United States or NYSDOH when they were announced, and when the claims against the last of the non-governmental defendants were discontinued on March 5, 1987, the jury empaneled for the determination of the claims against the non-governmental parties was discharged. Trial of plaintiff Jerome Andrulonis' claims against the United States and the Government's third-party claim against NYSDOH continued before the court.

Following the close of proof, an order was sought authorizing Joanna Andrulonis, as conservator of her husband's property, to settle Jerome's claims against the non-governmental parties and execute the necessary releases,[652] and an order was signed by the court on April 1, 1987. The United States then moved to vacate the order, which effectively approved the settlement arrangements previously announced. The Government argues that the allocation of settlement monies "seriously and unfairly

652. Rule 36 of the General Rules of the United States District Court for the Northern District of New York provides that "[a]n action by or on behalf of an infant or incompetent shall not be settled or compromised, or voluntarily discontinued, dismissed or terminated, without leave of the court embodied in an order, judgment or decree." *Compare* N.Y.C.P.L.R. § 1207 (McKinney Supp.1988); N.Y.C.P.L.R. Rule 1208 (McKinney 1976).

prejudices the rights and interests of the United States" under § 15–108 of New York's General Obligations Law, which defines and limits the rights of tortfeasors to contribution from other tortfeasors who may be liable for the same injuries suffered by a plaintiff who brings a personal injury action.

1. The "Good Faith" Requirement

Under Article 14 of New York's Civil Practice Law and Rules, a tortfeasor is entitled to contribution from another "subject to liability for damages for the same personal injury, injury to property or wrongful death," N.Y. C.P.L.R. § 1401 (McKinney 1976), in the amount of "the excess paid by him over and above his equitable share of the judgment recovered by the injured party," provided that the joint tortfeasor is not required to "contribute an amount greater than his equitable share" as a result of a contribution claim. N.Y. C.P.L.R. § 1402 (McKinney 1976). Article 14 provides that "[t]he equitable shares shall be determined in accordance with the relative culpability of each person liable for contribution." *Id.* Standing alone, the provisions of Article 14 would discourage settlement in cases involving more than one wrongdoer, since a settling defendant would remain potentially liable to other wrongdoers in subsequent suits, thereby depriving him of the assurance that he has "bought his peace." Consequently, § 15–108 of the General Obli-

gations Law was enacted in order to "clearly defin[e]" the effect a settlement with an injured party would have "on collateral rights and liabilities in future litigation." *Rock v. Reed-Prentice Division*, 39 N.Y.2d 34, 41, 382 N.Y.S.2d 720, 723, 346 N.E.2d 520, 523 (1976).

Section 15–108 provides as follows:

(a) Effect of release of or covenant not to sue tortfeasors. When a release or a covenant not to sue or not to enforce a judgment is given to one of two or more persons liable or claimed to be liable in tort for the same injury, or the same wrongful death, it does not discharge any of the other tortfeasors from liability for the injury or wrongful death unless its terms expressly so provide, but it reduces the claim of the releasor against the other tortfeasors to the extent of any amount stipulated by the release or the covenant, or in the amount of the consideration paid for it, or in the amount of the released tortfeasor's equitable share of the damages under article fourteen of the civil practice law and rules, whichever is the greatest.

(b) Release of tortfeasor. A release given in good faith by the injured person to one tortfeasor as provided in subdivision (a) relieves him from liability to any other person for contribution as provided in article fourteen of the civil practice law and rules.

(c) Waiver of contribution. A tortfeasor who has obtained his own release

> from liability shall not be entitled to contribution from any other person.

N.Y.Gen.Oblig.Law § 15–108 (McKinney 1978). The "right of set-off" provided by subdivision (a) "is intended to avoid a result in which the nonsettling tort-feasors bear more than their equitable share of the plaintiff's damages." *Lambert Houses Redevelopment Co. v. HRH Equity Corp.*, 117 A.D.2d 227, 232–33, 502 N.Y.S.2d 433, 436 (1st Dept.1986). Relying on *Hill v. St. Clare's Hospital*, 67 N.Y.2d 72, 499 N.Y.S.2d 904, 490 N.E.2d 823 (1986), and *Casey v. State*, 119 A.D.2d 363, 507 N.Y.S.2d 159 (2d Dept.1986), the United States contends that the settlements achieved in the case at bar undermine this purpose and that the court must set them aside or adjust the apportionment of the settlement funds to reflect the relative injuries suffered by Jerome and Joanna Andrulonis. The court finds that under the circumstances of this case, New York law does not require the modification of the settlement allocation suggested by the Government.

[94] In *Hill v. St. Clare's Hospital*, an individual suffered injuries resulting from the negligence of three original tortfeasors which were aggravated by the subsequent malpractice of a treating physician.[653] Un-

653. The terms of New York's comparative negligence statutes, and particularly of § 15–108, apply to successive as well as joint tortfeasors. *Hill v. St. Clare's Hospital*, 67 N.Y.2d at 83, 499 N.Y.S.2d at 911, 490 N.E.2d at 830.

der New York law, the original tortfeasors were liable for both the injuries traceable to their negligent acts as well as the injuries attributable to the subsequent malpractice. The injured individual brought separate actions against the original tortfeasors and the malpracticing tortfeasors and derivative claims were asserted in both cases by the injured individual's spouse. The action against the original tortfeasors was settled for a lump sum, and the plaintiffs executed releases to those defendants that neither distinguished between the main and derivative claims nor between the damages directly attributable to the original acts of negligence and the damages caused by the medical malpractice. 67 N.Y.2d at 75–77, 499 N.Y.S.2d at 906–07, 490 N.E.2d at 825–26. In the action against the parties charged with the ensuing malpractice, the defendants sought to interpose the settlement as a set-off to the plaintiffs' recovery. The trial court held that the defendants had the burden of proving which claims were covered by the settlement agreement and what proportion of the settlement sum designated in the release was to be assigned to each claim. *Id.* at 77–78, 499 N.Y.S.2d at 907, 490 N.E.2d at 826. The intermediate appellate court affirmed, but New York's Court of Appeals reversed, holding that N.Y.Gen. Oblig.Law § 15–108(a) "imposes upon the plaintiff who releases the original tort-feasor the burden of proving the extent to which his release reduces his claim against

a hospital or physician who through malpractice aggravates the original injuries." *Id.* at 75, 499 N.Y.S.2d at 906, 490 N.E.2d at 825. [654]

The holding in *Hill* is distinguishable from the case at bar, since in the present case there is no ambiguity concerning the terms of the settlements or the effect of the releases given to the private defendants. In reaching the result it did, however, the Court of Appeals, referring to the good faith requirement contained in N.Y. Gen.Oblig.Law § 15–108(b), found that "[t]o the extent that the injured party and the original tort-feasor are permitted to

654. The Court of Appeals noted that while the set-off provided by § 15–108(a) was an affirmative defense which must be pleaded by the party seeking to benefit from it, *see* N.Y.C.P.L.R. § 3018(b) (McKinney 1974 & Supp.1988), "the burden of proof is not a necessary concomitant of the burden of pleading." 67 N.Y.2d at 84, 499 N.Y.S.2d at 911, 490 N.E.2d at 830. Alluding to "the practicalities of the situation and ... a realistic evaluation of the relative positions of the parties" and observing that "it is the injured person who participates in and can control the fashioning of the terms of the settlement with the original wrongdoer, a procedure in which the successive wrongdoer has no part," *id.*, 499 N.Y.S.2d at 911, 490 N.E.2d at 830 (internal quotations omitted), the court concluded that the settling plaintiff had the burden of establishing the effect of the settlement agreement on the remaining action. 67 N.Y.2d at 84–86, 499 N.Y.S.2d at 911–12, 490 N.E.2d at 830–31.

stipulate the amount by which the liability of the successive tort-feasor is to be reduced by the original tort-feasor's payment, [the] intent [of the settling parties at the time of settlement] remains a factor, but the amount stipulated must also be shown to have been arrived at in good faith." 67 N.Y.2d at 84–85, 499 N.Y.S.2d at 912, 490 N.E.2d at 831. The case was remanded to the trier of fact for the purpose of conducting a hearing to determine the allocation of the total settlement amount between the two plaintiffs and the two sources of injury. The trial court was directed to "consider the statement as to allocation, if any, between original and aggravated injuries in the settlement documents and the gravity of the respective injuries and determine whether the amount allocated by the parties was arrived at in good faith." *Id.* at 86, 499 N.Y.S.2d at 912, 490 N.E.2d at 831.

In *Casey v. State*, decided shortly after the *Hill* decision was rendered, the Appellate Division for the Second Department was faced with a case in which separate claims for conscious pain and suffering and wrongful death were settled against two of three joint tortfeasors. Under New York law, recovery for a decedent's conscious pain and suffering accrues to the benefit of the decedent's estate, while damages recovered for wrongful death are personal to the "'distributees' of the decedent who have suffered 'pecuniary injury.'" *Casey*, 119 A.D.2d at 366, 507 N.Y.S.2d at 162 (quoting

Ratka v. St. Francis Hospital, 44 N.Y.2d 604, 609, 407 N.Y.S.2d 458, 460, 378 N.E.2d 1027, 1029 (1978)). Consequently, in one respect *Casey*'s procedural posture was akin to the case at bar: two claimants (in *Casey*, two groups of claimants) with causes arising out of the same series of negligent acts settled their claims with some but not all of the alleged joint tortfeasors claimed to be responsible for the separate and distinct injuries suffered.

In *Casey*, the settling parties had failed to allocate the settlement between the wrongful death and pain and suffering causes of action, but subsequently a petition for allocation of the settlement monies between the pain and suffering claims and the wrongful death claims was submitted to and approved by New York's Surrogate's Court (this was done for estate and taxation purposes). The nonsettling tortfeasor (in this case, the State of New York) then moved to assert as an affirmative defense to the action that had been brought against it in New York's Court of Claims a right to set-off in the entire amount of the settlement. The claimants argued that the State was bound by the apportionment contained in the decree issued by the Surrogate's Court. The Court of Claims ruled that it would make a determination, independent of the agreement of the settling parties and the decree of the Surrogate's Court, of the proper allocation of the settlement funds for the purpose of determining

the set-off under § 15–108 after trial. The Second Department affirmed. 119 A.D.2d at 365, 507 N.Y.S.2d at 161.

The appellate court framed the issue raised as whether "the settling parties themselves make the apportionment which would be binding on a nonsettling tortfeasor." *Id.* at 366, 507 N.Y.S.2d at 162. The Second Department concluded that "any such settlement sum, even though allocated by the parties or by a decree of the Surrogate, must, for the purpose of the actual setoff, be apportioned at trial...." *Id.*, 507 N.Y.S.2d at 162.

> We hold that the self-created apportionment proffered by the claimants is not binding on the trial court. We are dealing with two separate and distinct causes of action. The State is entitled to a true setoff based on the merits of the respective causes of action. This apportionment must be predicated on the evidence adduced at the trial with regard to the monetary value of each cause of action.

Id. at 367, 507 N.Y.S.2d at 163.

Casey is factually distinguishable from the case at bar, since it too involved a settlement for distinct claims in a gross unallocated amount, although the settling parties did attempt to clarify their intentions after the fact. One court has interpreted *Hill* to stand for the proposition that the burden is on the plaintiff "to establish why and to what extent [a settlement] should be accorded less than its ap-

parent effect." *Carter v. State*, 139 Misc.2d 423, 428–29, 528 N.Y.S.2d 292, 296 (Ct.Cl.1988). Impliedly, if the allocation of settlement monies are unambiguously set forth in the settlement agreement, there is no possibility that adherence to that allocation would result in according the settlement "less than its apparent effect." If the allocation does not fairly reflect the relative culpability of the parties, it would seem that the provision in N.Y.Gen. Oblig.Law § 15–108(a) allowing reduction of an award by the percentage of a settling tortfeasor's "equitable share of the damages" would adequately protect the nonsettling tortfeasors.

In the present case, the intention of the settling parties was clear from the terms of the settlement agreements that were placed on the record, and if the interpretation of *Hill* and *Casey* offered above were followed, the court would not have to inquire into whether the apportionment in those settlements were devised in "good faith." Nonetheless, there is broad language in both *Hill* and *Casey* supporting the Government's contention that a trial court must independently assess the reasonableness of the apportionment of settlement funds even when the intent of the parties at the time of settlement is unambiguous, at least when determining the effect that settlement will have on the setoffs allowed nonsettling tortfeasors under § 15–108. In *Hill*, the Court of Appeals indicated that in determining the allocation

of settlement monies between original and aggravated injuries, statements concerning such allocation contained in the settlement agreement itself would not be conclusive. 67 N.Y.2d at 86, 499 N.Y.S.2d at 912, 490 N.E.2d at 831. *Casey* states that the apportionment of monies between separate and distinct causes of action for the purpose of determining a "true" set-off must be based on the relative merits of the two causes of action. 119 A.D.2d at 367, 507 N.Y.S.2d at 163. For the purposes of resolving the Government's motion, the court will assume that it must independently ratify the apportionment of settlement funds for purposes of determining the Government's set-off under § 15–108.

The United States appears to interpret *Hill* and *Casey* to mean that if the apportionment of monies used to settle a primary claim and a derivative claim do not closely approximate the relative injuries suffered by the two settling plaintiffs, the settlement must be set aside. Noting that the injuries suffered by Jerome Andrulonis exceed those suffered by Joanna in severity, the Government asserts that the court order approving the settlement arrangement between plaintiffs and the non-governmental defendants must be vacated.

[95] Initially, the court notes that the cases relied upon by the Government do not support vacatur of the court's order approving of the settlement; at best, they require the court to independently assess

the reasonableness of the apportionment of the settlement monies and, if appropriate, adjust that apportionment in determining the set-off to which the nonsettling defendants are entitled under § 15–108. As stated by the Second Department, "[i]t is firmly established that a valid release which is clear and unambiguous on its face and which is knowingly and voluntarily entered into will be enforced as a private agreement between parties." *Appel v. Ford Motor Co.*, 111 A.D.2d 731, 732, 490 N.Y.S.2d 228, 229 (2d Dept.1985). As between plaintiffs and the settling defendants, the settlement agreement and the releases given are binding.[655]

655. In the papers filed supporting its motion to vacate the settlement agreements, the Government contends that by apportioning the settlement fund as they did, Joanna Andrulonis and her attorneys "exploited" the interests of Jerome. There is no little irony in this assertion given the questions raised concerning the conduct of the attorney for the Justice Department who represented the Government at one point in this litigation. *See* text, *infra* at 1536–37. The Government's new-found concern for the welfare of Jerome Andrulonis is just as transparent as the plaintiffs' motivation for apportioning the settlement monies as they did. Plaintiffs wish to minimize the set-off provided by § 15–108 and thereby maximize the recovery ultimately obtained by Jerome Andrulonis, and the Government wishes to maximize its set-off and minimize its liability. To imply at this stage of the litigation that the Government's purpose in opposing the settlement is somehow more "pure" than the motivations of Joanna

[96] Moreover, the court rejects the Government's suggestion that *Hill* and *Casey* require that the apportionment of a settlement fund used to compromise the claims of more than one plaintiff must in every instance approximate the relative injuries of the plaintiffs for the purpose of calculating the § 15–108 set-off for a non-settling defendant. The Government ignores the fact that as a result of the settlement agreements and the procedural posture of this case, the non-governmental defendants will bear the entire cost of the damages suffered by Joanna Andrulonis, including that portion for which the negligence of the United States and the New York State Department of Health are re-

Andrulonis and her attorneys in fashioning it is insulting, particularly in light of the history of this case. Throughout this lawsuit, the Government has been obstructive, unnecessarily escalating the cost and complexity of this litigation. There is cause for suspicion that some Government agent, possibly an attorney for the Justice Department, has withheld evidence from the other parties to this lawsuit. In contrast to the Machiavellian conduct of the United States, Joanna Andrulonis has labored to provide her husband with the best life possible, under conditions which understandably would compel a less determined person to resort to the institutionalization of the victim of this tragic accident. In light of the loyalty, strength and love exhibited by her efforts, the Government's suggestion that Joanna Andrulonis would "exploit" her husband's interests is galling.

sponsible. Joanna's derivative claim against the United States was dismissed earlier in this litigation because she failed to timely file an administrative claim on her own behalf, a jurisdictional prerequisite to asserting a claim against the Government under the FTCA. 28 U.S.C. §§ 2401, 2675; *see Andrulonis v. United States*, No. 79-CV-847 (N.D.N.Y. March 8, 1984). New York's Workers' Compensation Act precludes Joanna from asserting a tort claim against the State. *See* N.Y.Work. Comp.Act §§ 11, 29(6) (McKinney Supp. 1989). Although the non-governmental defendants asserted cross-claims against the United States under the FTCA for contribution and could have asserted similar claims against the State in New York's Court of Claims had Joanna prevailed in this action, those claims were waived when they entered into their settlements with plaintiffs. N.Y.Gen.Oblig.Law § 15-108(c). Consequently, the amount for which Joanna's claims were settled represents the entirety of the damages she will recover, and neither the Government nor the State will be required to contribute to this sum. In contrast, the amount for which Jerome's claims against the non-governmental defendants were settled does not exhaust the remedies available to him.

[97] Under these circumstances, the court believes that there are two distinct inquiries to be made in assessing whether the releases at issue were executed in good faith. First, the court must determine

whether the amount set aside for the primary claims of Jerome Andrulonis—$225,000—was a reasonable estimate of the settling defendants' equitable share of the total damages he suffered. Second, the court must determine whether the amount set aside for the derivative claims of Joanna Andrulonis—$1,075,000—was unreasonably excessive in light of the damages she actually suffered. The court believes that these questions must be answered by reference to an objective standard of "good faith;" the primary concern of the New York courts in this area appears to be the assurance that the apportionment of settlement funds are reasonable. The motivation of the parties in allocating settlement funds as they do seems irrelevant.[656]

2. Application of the "Good Faith" Requirement

[98] Turning to the first inquiry set out in the paragraph above, the court believes that the amount directed toward the primary claims of Jerome Andrulonis against the non-governmental parties did not grossly underestimate the settling parties' eq-

656. In any event, the subjective motivation of plaintiffs is obvious: they want to maximize any possible recovery against the Government by allotting as little of the settlement funds to Jerome's claims as possible.

uitable share of the damages he suffered. There is a noticeable discrepancy between the amount Lilly and Thompson & Sons settled for—$100,000—and the amount that the court found was the equitable share of the damages suffered by Jerome Andrulonis for which those two defendants were responsible—$321,232. However, the total amount Jerome received through all of the settlement agreements—$225,000—is not so far off the total amount ultimately apportioned to those defendants—again, $321,232—that it raises an inference of bad faith. It is appropriate to look at all of the settling defendants as a group in this case, since plaintiffs had weak cases against all of those defendants, and may have been motivated by the desire to recover as much as they could without much consideration for the relative culpability of any of them.

[99] The second question is whether the $1,075,000 allocated to Joanna Andrulonis' derivative claims is reasonable. In *Millington v. Southeastern Elevator Corp.*, 22 N.Y.2d 498, 293 N.Y.S.2d 305, 239 N.E.2d 897 (1968), the New York Court of Appeals recognized the common law right of a woman to maintain a cause of action for loss of consortium against a tortfeasor who has

caused her husband personal injury.[657] *Id.* at 509, 293 N.Y.S.2d at 313, 239 N.E.2d at 905. Although a consortium claim is deemed a separate interest personal to the wife, *Haspil v. Church of St. Cyril*, 128 Misc.2d 968, 970–71, 491 N.Y.S.2d 914, 917 (Sup.Ct.1985), it is derivative in the sense that the claim does not exist "independent of the injured spouse's right to maintain an action for injuries sustained." *Liff v. Schildkrout*, 49 N.Y.2d 622, 632, 427 N.Y.S.2d 746, 749, 404 N.E.2d 1288, 1291 (1980). Thus, a defendant who has not been found to be a tortfeasor who caused physical or mental injury to the husband cannot be held liable for the loss of consortium suffered by the wife as a result of her husband's injuries. *See Belanoff v. Grayson*, 98 A.D.2d 353, 358, 471 N.Y.S.2d 91, 94 (1st Dept.1984); *Maidman v. Stagg*, 82 A.D.2d 299, 301, 441 N.Y.S.2d 711, 713 (2d Dept. 1981).

657. Prior to *Millington*, New York's common law permitted only a husband to maintain an action for loss of consortium. The *Millington* decision "terminat[ed] an unjust discrimination under New York law" by allowing consortium claims by either spouse. 22 N.Y.2d at 509, 293 N.Y.S.2d at 313, 239 N.E.2d at 905. Because the consortium claim of Joanna Andrulonis is at issue in the case at bar, the remainder of the court's discussion will speak exclusively in terms of a wife's cause of action for loss of consortium.

[100, 101] A consortium claim is rooted in the interest of an injured party's spouse "in the continuance of a healthy and happy marital life," *Millington*, 22 N.Y.2d at 504–05, 293 N.Y.S.2d at 309–10, 239 N.E.2d at 901–02, and consequently the assessment of damages for loss of consortium must be based on "the real injury done to the marital relationship." *Id.* at 504, 293 N.Y.S.2d at 309, 239 N.E.2d at 901. Under New York law, this injury to the marriage relationship can involve both tangible and intangible elements. *Id.* at 502, 293 N.Y.S.2d at 308, 239 N.E.2d at 900; *see* Annot., *Wife's Loss of Consortium—Damages*, 74 A.L.R.3d 805, 809 (1976). Under the passionless designation of "loss of services," the law has recognized that a variety of tangible harms suffered by the wife as a result of her husband's physical injury that may be redressed by monetary damages. For example, if it is shown that as a result of her husband's injury the wife will be required to assume greater responsibility for normal household duties or yard work, she is entitled to recover damages. *Wright v. State*, 110 A.D.2d 1060, 1061, 488 N.Y.S.2d 917, 919 (4th Dept.), *aff'd*, 66 N.Y.2d 452, 497 N.Y.S.2d 880, 488 N.E.2d 810 (1985); *Good v. Mantaibano*, 50 A.D.2d 885, 885, 377 N.Y.S.2d 167, 168 (2d Dept. 1975). If the evidence establishes that the wife must assume the burden of rearing her children alone when before her husband's injury both parents were involved in this endeavor, the wife has suffered a

tangible injury compensable under New York law. *See Rocha v. State*, 77 Misc.2d 290, 301, 352 N.Y.S.2d 990, 1003 (Ct.Cl.), *aff'd*, 45 A.D.2d 633, 360 N.Y.S.2d 484 (3d Dept.1974).

[102] A wife can also recover for the often more substantial intangible injuries she incurs when her husband suffers debilitating physical or mental injuries. The intangible aspects of consortium include "such elements as love, companionship, affection, society, sexual relations, solace and more." *Millington*, 22 N.Y.2d at 502, 293 N.Y.S.2d at 308, 239 N.E.2d at 900.

> The loss of companionship, emotional support, love, felicity and sexual relations are real injuries. The trauma of having to care for a permanent invalid is known to have caused mental illness. There may not be a deterioration in the marital relationship, but it will certainly alter it in a tragic way.

Id. at 503, 293 N.Y.S.2d at 308, 239 N.E.2d at 900. In assessing the intangible elements of the wife's loss, a court must consider the nature and happiness of the marriage prior to the husband's injury, *see Christman v. Bailey*, 38 A.D.2d 773, 327 N.Y.S.2d 966 (3d Dept.1972), how the marital relationship has changed subsequent to the husband's injury, whether that change is attributable in whole or in part to any material change in the husband's personality, attitudes, and capabilities, the causal relationship between the husband's injury

and any change in his personality and capabilities, and the permanency of these changes in the husband. Annot., *Wife's Loss of Consortium—Damages*, 74 A.L.R.3d at 811.

Even though the settlements reached by plaintiff Joanna Andrulonis removed any incentive she had to put in all of her proof regarding her consortium claims, the record is filled with evidence that the effect her husband's injuries have had on her marriage has been extraordinary. Joanna has taken primary responsibility for the day-to-day care of her husband ever since his release from AMCH in March 1978, while working full time as a teacher to support her family. The task of caring for Jerome Andrulonis can be all-consuming; he cannot initiate self-care activities, and his lack of safety consciousness requires his constant surveillance. His size and strength coupled with recurrent periods of agitation make it difficult to control him. Jerome remains prone to strike out at his wife suddenly and unexpectedly; there have been periods when his violent outbursts were frequent.

Because of the nature of her husband's injuries, Joanna was left with the responsibility of raising her family alone. Jerome has not contributed to housework or yardwork since his illness. Limitations have been imposed on a wide range of social activities in which Joanna previously engaged, from attending church services to dining out at restaurants to patronizing

movie theaters. Jerome is unable to communicate with Joanna or their family in any meaningful way; Joanna has permanently lost the counsel and guidance of her husband; it is clear that Jerome cannot express the love or provide the society and companionship that by all indications characterized what was before April 1977 a happy and successful marriage. The material personality and behavioral changes in Jerome Andrulonis attributable to his illness are permanent.

Evidence of the injury Joanna has suffered can be found in the passing comments she and others made while testifying at trial. A neurologist who treated Jerome mentioned that his patient's constant drooling and inability to control the facial muscles near his mouth troubled Joanna deeply. Joanna noted that when Jerome was admitted into AMCH's psychiatric ward in 1979, she would not allow her children to visit him because it was "a very difficult ward;" [658] as she said this, it was evident that after eight years it still pained her that she had been compelled to leave her husband there for four months. Joanna continues to maintain an unrealistically optimistic belief that her husband will regain

658. Tr. at 1735 (Joanna Andrulonis).

some of his cognitive abilities; it is telling that Joanna preferred to leave the courtroom whenever medical testimony regarding her husband's condition was offered.

In determining whether the total amount received by Joanna Andrulonis in settlement—$1,075,000—was a good faith assessment of the value of her claims, the court's inquiry is guided by the standard applied when the reduction of the damages awarded by a jury is sought. A jury verdict "must be set aside where it is so disproportionate to the injury suffered 'as to shock the judicial conscience.'" *Attridge v. Cencorp Division of Dover Technologies International, Inc.*, 836 F.2d 113, 117 (2d Cir.1987) (citation omitted); *see also Martell v. Boardwalk Enterprises, Inc.*, 748 F.2d 740, 750 (2d Cir.1984). It is appropriate to adopt the same standard in assessing whether a settlement figure is unreasonably excessive, since by settling the parties forswore their right to have a jury determine Joanna Andrulonis' consortium claims. As in cases in which jury verdicts are attacked, the court will seek guidance from other awards countenanced by courts in somewhat similar cases, but will be mindful that such "comparative analysis" is not determinative. *Attridge*, 836 F.2d at 117–18 (citing *Batchkowsky v. Penn Central Co.*, 525 F.2d 1121, 1124–25 (2d Cir. 1975)); *see also Senko v. Fonda*, 53 A.D.2d 638, 639, 384 N.Y.S.2d 849, 851 (2d Dept. 1976) (prior awards "may guide and enlighten the court[s]" in determining whether jury award was excessive).

[103] The injury to Joanna Andrulonis' marital relationship has been acute, and if her claims had gone to trial and liability on the part of the settling non-governmental parties had been shown, it would be reasonable to expect that her recovery would likely to have been among the highest awards for loss of consortium reported in this state. In the comparable case of *McDougald v. Garber*, 132 Misc.2d 457, 504 N.Y.S.2d 383 (S.Ct.1986), *aff'd*, 135 A.D.2d 80, 524 N.Y.S.2d 192 (1st Dept.1988), *modified and remanded on other grounds*, 73 N.Y.2d 246, 538 N.Y.S.2d 937, 536 N.E.2d 372 (1989), the court found that a jury award for $1,500,000 was "amply supported by the record." 132 Misc.2d at 462–63, 504 N.Y.S.2d at 387. In *McDougald*, a thirty-one-year-old woman suffered a severe brain injury as the result of oxygen deprivation during a surgical procedure in 1978. The woman lapsed into a comatose state in which she had remained at the time of trial. She had been confined to the hospital since the date of her injury. The neurological injury she had suffered was permanent. The trial court noted that

> [t]he impact of the injury inflicted on his wife on Mr. McDougald's life is, indeed, substantially greater than had Mrs. McDougald died as a result of her injuries. He is not only left with the task of raising his two children and maintaining his household ... but in addition is a constant attendant at the bedside of his wife. Proof established that he spends

> approximately three hours a day, almost every day, with her and there is little doubt, based on the relationship between Mr. and Mrs. McDougald demonstrated at trial, that such vigilant attendance will continue as long as they both live.

Id. at 463, 504 N.Y.S.2d at 387. The dedication shown by Joanna Andrulonis to her husband is akin to that of the husband in *McDougald*, and the burdens placed on Joanna by her husband's injury are arguably greater.

There are examples of other cases in other jurisdictions involving serious injury to the marital relationship in which awards were approved that are comparable to the amount for which Joanna Andrulonis' claims were settled. Those cases are discussed in the margin.[659] In light of this

659. In *Harrigan v. Ford Motor Co.*, 159 Mich. App. 776, 406 N.W.2d 917 (C.A.), *appeal denied*, 429 Mich. 887 (1987), an award of $1,000,000 for loss of consortium to a woman whose husband was rendered a quadriplegic in an automobile accident was found not excessive. The court awarded $1,800,000 for loss of consortium resulting from wrongful death (a cause of action recognized by Texas law) in *Wheat v. United States*, 630 F.Supp. 699 (W.D.Tex.1986). In that case, a succession of physicians at an United States Army hospital negligently failed to diagnose a woman's cervical cancer. The court observed that her husband watched his wife "slowly die a lingering death" without understanding the cause of her physical ailments. In

authority, the court concludes that the settlement monies apportioned to the derivative claims were not so excessive as to "shock the judicial conscience."

a 1978 case, *Rodriguez v. McDonnell Douglas Corp.*, 87 Cal.App.3d 626, 151 Cal.Rptr. 399 (2d Dist.1978), the court approved a jury award of $500,000 to a twenty-year-old woman for injury to the marital relationship resulting from her twenty-two-year-old husband's permanent disability as a result of an industrial accident. At the time of the accident, the couple had been married for only sixteen months, but the evidence indicated that the woman remained devoted to her husband. The husband had severe permanent injuries, including loss of bowel and bladder function, leaving him dependent on enemas and laxatives for bowel function, loss of sexual function, constant pain in his left arm, surgical removal of a portion of his stomach, and uncontrolled spasms. The husband could not dress or bathe himself, would require the care of specialists for the rest of his life, and was expected to live out his normal life expectancy. The physical and psychological demands on the wife were monumental. In a 1977 case, *Ossenfort v. Associated Milk Producers, Inc.*, 254 N.W.2d 672 (Minn.1977), a $500,000 award was not deemed excessive in a case where a husband suffered anatomical brain damage and was rendered a spastic quadriplegic as a result of a truck collision. In *General Electric Co. v. Bush*, 88 Nev. 360, 498 P.2d 366 (1972), a $500,000 award for loss of consortium was found not to be excessive. In that case, the plaintiff's husband was paralyzed from the neck down, was unable to communicate or care for himself, and suffered some loss of mental function.

In sum, the court finds that the apportionment of settlement monies was made in good faith, given the procedural posture of this case. This is not a case where plaintiffs released one tortfeasor for a nominal sum in return for a promise of cooperation in any attempt to extract more from the remaining tortfeasors than their equitable share of the damages. *See Friend v. Dibble*, 124 Misc.2d 151, 153, 475 N.Y.S.2d 765, 766 (Sup.Ct.1984). The United States has not been prejudiced by the arrangement, since under § 15–108 "settlement by one tortfeasor always benefits the remaining tortfeasors by reducing their liability at least by the amount of the settlor's equitable share...." *Id.* at 154, 475 N.Y.S.2d at 767 (quotation omitted). The settlement disadvantaged the United States in that after it, plaintiffs no longer had the incentive to prove that the settling parties were in any way responsible for the injuries that Jerome Andrulonis sustained, and thus the burden of putting into evidence the case against the settling defendants for all practical purposes shifted to the Government and the State. But this is the result every time some but not all of the joint tortfeasors responsible for a plaintiff's injuries reaches a settlement with that plaintiff. If this were a ground for disregarding the terms of the settlement in applying § 15–108, injured parties would lose much of their incentive to settle claims against joint tortfeasors, and the entire purpose of § 15–108 would be undermined. *See Rock v. Reed-Prentice*, 39 N.Y.2d at 41, 382 N.Y.S.2d at 723, 346 N.E.2d at 523.

B. *Sanctions*

As the trial of this matter neared its close, the Government attempted to introduce through Dr. Baer a document recording the manner in which Baer had prepared the ERA-BHK/21 virus strain he brought to Albany on March 28, 1977 (hereinafter "the passage history document").[660] This document had been the subject of interrogatories propounded by plaintiffs and directed at the Government in 1980 and had been requested by plaintiffs through a formal notice for production of documents in 1981. Other parties to this action inquired about the existence of such a document as well. In its answers to plaintiffs' interrogatories, the Government indicated that there were no "worksheets, memoranda, [or] reports dealing with the preparation of the virulent rabies virus" transported to Griffin by Dr. Baer in March 1977.[661] The Government again denied the existence of any documents detailing the history of the virus strain that caused Andrulonis' disease in its response to the notice of production in 1981.[662] When the Government attempted

660. Court Exh. 1.

661. Doc. 135, No. 27(i).

662. Doc. 180, Response No. 2.

to question Dr. Baer concerning the passage history document five weeks after the trial had commenced, plaintiffs objected, and the court excluded the document under Fed.R.Civ.P. 37(d). Because neither plaintiffs nor the State had access to the document nor were able to use it either in questioning plaintiffs' expert witnesses or in adequately preparing for the cross-examination of Dr. Baer, the court deemed exclusion an appropriate sanction.

Plaintiffs present strong arguments in support of even harsher sanctions. They note that while the initial reports concerning the Andrulonis case indicated that the titer of the virus used on March 29, 1977 was 8.5,[663] the Government's answers to the interrogatories propounded in 1980 indicated the titer of the virus was 8.1, and that the deposition testimony of Dr. Baer in 1981 was to the same effect. Among other things, the passage history document indicates that the titer of that virus strain was indeed 8.1. Plaintiffs suggest that there is reason to believe that some Government agent—impliedly Baer or Justice Department attorney Faith Burton, Esq., or both—knew of the existence of the passage history document and withheld it from the other parties involved in this case.

663. *See* "Rabies in New York Laboratory Worker," *supra* note 4, at 183; NYS Retirement System Report, *supra* note 306.

The Government has submitted evidence that Baer had sent a photocopy of the passage history document to Burton in 1982, and submits that a search of the Government's files failed to recover that photocopy.[664] The Government suggests that the photocopy either was misplaced or was lost before reaching Burton. The Government further submits that the document fails to reveal relevant information not otherwise made available to the other parties to this lawsuit, and thus did not prejudice plaintiffs or any of the other parties. The Government's explanation is plausible and if the controversy surrounding this document was an isolated incident in this lawsuit, the court might be willing to accept the Government's explanation on face value.

The failure to produce the passage history document was not an isolated controversy, however. Discovery during the course of this lawsuit was particularly difficult, characterized by rancorous disputes and a lack of cooperativeness on the part of most of the parties. During the course of the pretrial proceedings in this lawsuit, particularly in the period in which attorney Burton

664. *See* Doc. 608, especially Exh. 1 attached thereto.

represented the United States, a number of disturbing events transpired. Most disturbing to the court is the deposition testimony of a number of Government agents. During these depositions, the agents deposed consistently gave vague, unresponsive, or misleading answers to simple, straightforward questions.[665] Plaintiffs note that after attorney Burton assumed control of this case, an amended answer was filed by the Government directed at allegations in plaintiffs' amended complaint identical to those contained in the original complaint, and the amended answer denied allegations that were admitted in the original answer. The Government points out that some of the changes made were warranted by the facts established at trial; in many instances, however, they were not. The Government's refusal to concede in its amended answer points it had admitted in its original answer may well have escalated the costs of this lawsuit.

[104] Plaintiffs have moved for sanctions under Rule 11, Fed.R.Civ.P., 28 U.S.C. § 1927, and the inherent power of the court. The court finds that it has jurisdic-

665. Some examples of the evasive and misleading testimony given by these Government agents during their depositions are in evidence. *See* Tr. at 873-936 (Winkler Deposition); Tr. at 1109-1222 (Baer Deposition); Exh. G-125I (Mallison Deposition).

tion to consider this motion, notwithstanding the limitations of sovereign immunity. *See, e.g., United States v. Gavilan Joint Community College Dist.*, 849 F.2d 1246, 1251 (9th Cir.1988); *Joseph v. United States*, 121 F.R.D. 406, 413–14 (D. Hawaii 1988); *Larkin v. Heckler*, 584 F.Supp. 512 (N.D.Cal.1984); *cf. Adamson v. Bowen*, 855 F.2d 668 (10th Cir.1988) (28 U.S.C. § 2412 waives the government's sovereign immunity as to Rule 11 sanctions). Imposition of sanctions in this case, regardless of the authority used, requires a "clear showing of bad faith." *Oliveri v. Thompson*, 803 F.2d 1265, 1273 (2d Cir.1986) (quotation omitted) (discussing § 1927), *cert. denied*, 480 U.S. 918, 107 S.Ct. 1373, 94 L.Ed.2d 689 (1987); *id.* at 1272 (discussing court's inherent power); *Nemeroff v. Abelson*, 620 F.2d 339 (2d Cir.1980) (discussing standard used before Rule 11 was amended in 1983). To resolve this issue, which will require some inquiry into the subjective motivations of agents of the Government and of a Justice Department attorney, it will be necessary to conduct a hearing. The court directs the parties to appear at a conference to be held at the United States Courthouse in Albany, New York at 3:00 in the afternoon on November 27, 1989. At that conference, the court will entertain proposals concerning the manner in which the hearing should be conducted and the subject matter of that hearing.

VII. CONCLUSION

The Clerk of the Court is directed to enter judgment on behalf of plaintiff Jerome Andrulonis against defendant United States of America in the amount of $5,978,-409. The Clerk is further directed to enter judgment on behalf of third-party plaintiff United States of America against third-party defendant New York State Department of Health in the amount of $3,729,785.

It is So Ordered.

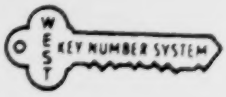
ORDER

The United States [the "government"] has moved for a new trial, or to vacate, alter or amend this court's judgment entered on October 18, 1989. With one exception the motion is denied. The court grants the motion only insofar as increasing the award to which third-party plaintiff United States is entitled from third-party defendant New York State Department of Health ["NYSDOH"]. In the Memorandum–Decision and Order filed in October 17, 1989, this court miscalculated the amount of contribution which the government was entitled to receive from NYSDOH. Properly calculated, the government is entitled to contribution from NYSDOH for 65/95ths, or 68.42%, of the award after deductions are taken from the total award to account for the liability of Eli Lilly and Co. and Thompson & Sons & Co., as well as the settlement entered into by the WARF defendants and the Glatt defen-

dants. The award, after making the above two deductions, is $5,978,409. Accordingly, the government is entitled to contribution from NYSDOH in the amount of 68.42% of $5,978,409, or $4,090,427.

The clerk of the court is directed to enter an amended judgment on the behalf of third-party plaintiff United States against third-party defendant New York State Department of Health in the amount of $4,090,427, not $3,729,785 as originally directed by this court.

It is so Ordered.



Opinion of United States District Court for the Northern District of New York, Dated October 3, 1984.

ANDRULONIS v. UNITED STATES

Cite as 593 F.Supp. 1336 (1984)

Joanna ANDRULONIS, as Conservator of the Property of Jerome Andrulonis, Plaintiff,

v.

UNITED STATES of America; Glatt Air Techniques, Inc.; Glatt GmbH (Germany); Wisconsin Alumni Research Foundation, Inc.; Warf Institute, Inc.; Raltech Scientific Services, Inc.; Ralston Purina Company; Eli Lilly and Company; and John L. Thompson and Sons and Company, Defendants.

UNITED STATES of America, Third-Party Plaintiff,

v.

NEW YORK STATE DEPARTMENT OF HEALTH, Third-Party Defendant.

No. 79-CV-847.

United States District Court, N.D. New York.

Oct. 3, 1984.

Negligence action was brought under Federal Tort Claims Act. Government moved to dismiss or for summary judgment. The District Court, Munson, Chief Judge, held that Government's employee's

negligence in carrying out his responsibilities with respect to development of method for orally immunizing wildlife against rabies was not subject to discretionary function exception of the Federal Tort Claims Act.

Motion denied.

Roemer & Featherstonhaugh, Albany, N.Y., for plaintiffs; Dean J. Higgins, Albany, N.Y., of counsel.

Anderson Russell Kill & Olick, P.C., New York City, Paul F. Donahue Associates, Albany, N.Y., for defendant Glatt GmbH (Germany); Irene C. Warshauer, Marcy Louise Kahn, Robert P. Reichman, New York City, of counsel.

Carter, Conboy, Bardwell, Case & Blackmore, Albany, N.Y., for defendant Wisconsin Alumni Research Foundation; Jeffrey J. Tymann, Albany, N.Y., of counsel.

McNamee, Lochner, Titus & Williams, Albany, N.Y., for defendants, Raltech Scientific Services, Inc. and Ralston Purina Co.; Paul Scanlan, Albany, N.Y., of counsel.

Maynard, O'Connor & Smith, Schenectady, N.Y., for defendant Warf Institute, Inc.; Richard Gershon, Schenectady, N.Y., of counsel.

Ainsworth, Sullivan, Tracy & Knauf, Albany, N.Y., for defendants Eli Lilly and Co. and John L. Thompson & Sons & Co.; Thomas F. Tracy, Albany, N.Y., of counsel.

Galef & Jacobs, New York City, for defendant Glatt Air Techniques, Inc.; Christopher M. Houlihan, New York City, of counsel.

Frederick J. Scullin, Jr., U.S. Atty., Robert Abrams, Atty. Gen. of the State of N.Y., Albany, N.Y., for defendant New York State Dept. of Health; William Fanciullo, Asst. U.S. Atty., Kevan J. Acton, Asst. Atty. Gen., Albany, N.Y., of counsel.

Dept. of Justice, Civ. Div., Torts Branch, Washington, D.C., for U.S.; Lizabeth A. McKibben, Trial Atty., Washington, D.C., of counsel.

ORDER

MUNSON, Chief Judge.

The defendant United States has renewed its Rule 12(b) motion to dismiss or for summary judgment under Rule 56 following the recent decision by the Supreme Court on the discretionary function exception to the Federal Tort Claims Act, 28 U.S.C. § 2680(a) (1982), [FTCA], *United States v. S.A. Empresa DeViacao Aerea Rio Grandense (Varig Airlines)*, — U.S. —, 104 S.Ct. 2755, 81 L.Ed.2d 660 (1984), [*Varig*]. The Government's previous motion to dismiss was denied on December 19, 1983. After careful review of the parties' memoranda and after oral arguments on the motion, the court concludes that the challenged governmental conduct in the case at bar does not fall within the discretionary function exception to the Federal Tort Claims Act.

In *Varig* the Supreme Court held that the discretionary function exception to the FTCA precluded a negligence action against the Federal Aviation Administration challenging the regulatory scheme for the promotion of airplane safety and the Government's selected method for implementing the scheme. Plaintiffs in *Varig* alleged negligence in the FAA's certification and particularly the "spot-checking" method selected for insuring compliance with the FAA's safety regulations which allegedly caused in-flight fires and resulting death and injuries. It is significant to note that the plaintiffs in *Varig* did not allege specific acts of negligence on the part of FAA employees in the actual inspection of the defective planes. Instead, the plaintiffs were challenging the effectiveness of the FAA's spot-checking system in insuring compliance with safety regulations. The Court held that a challenge to the method selected by the FAA in the exercise of its regulatory authority implicated policy decisions; these policy decisions and their implementation were the type of exercise of discretionary authority that Congress intended to immunize from tort liability under the discretionary function exception, 28 U.S.C. §-2680(a). *Varig*, — U.S. at ——, 104 S.Ct. at 2768.

In the instant case plaintiffs make specific allegations of negligence on the part of the Government's employee Dr. Baer in carrying out his responsibilities with re-

spect to the development of a method for orally immunizing wildlife against rabies. For the purposes of this motion the court will view the facts in the light most favorable to plaintiffs as the nonmoving parties, *see Project Release v. Prevost*, 722 F.2d 960, 968 (2d Cir.1983); *Patrick v. LeFevre*, 745 F.2d 153 at — (2d Cir.1984). Specific allegations of negligent conduct by Dr. Baer include: the production of the virus with an increased titer more hazardous to humans, failure to warn of the known hazards of the virus, written assurance given as to the safety of the virus and Dr. Baer's personal participation in the introduction of the virus into the "Uni-Glatt" machine when Dr. Baer knew or should have known that conditions at the Griffin Laboratory were unsafe. As a result of his exposure to the rabies virus at Griffin Laboratory, plaintiff developed rabies and suffered irreversible brain damage.

The specific allegations of negligence on the part of the individual governmental employee Dr. Baer are a far cry from the broad challenge asserted in *Varig* to the regulatory scheme developed by the FAA. None of the cases submitted by the Government which were decided subsequent to *Varig*, nor any case which the court has been able to locate independently, militate against adherence to this court's previous denial of the Government's motion to dismiss or for summary judgment.

The Government has presented five cases decided subsequent to *Varig* in which the discretionary function exception to the

FTCA was held to preclude negligence actions against the federal government. None of the cases present factual situations similar to the instant case. However, a discussion of these cases may be worthwhile.

National Gas Pipeline Co. v. United States, 742 F.2d 502 (9th Cir.1984), presented a challenge to FAA safety inspections similar to that in *Varig*. Relying directly on *Varig*, the Ninth Circuit dismissed the claim against the Government. Similarly, the Third Circuit has recently held that claims based on the negligent exercise of regulatory authority by the federal Nuclear Regulatory Commission are barred by the discretionary function exception. *General Public Utilities Corp. v. United States*, 745 F.2d 239 (3d Cir.1984).

In *Flammia v. United States*, 739 F.2d 202 (5th Cir.1984), the Fifth Circuit applied the discretionary function exception to preclude an action against the Immigration and Naturalization Service [INS] by a police officer wounded in a burglary committed by a Cuban refugee. To the extent that the plaintiff alleged that the INS should not have admitted the particular person into the United States or released him from the "processing center" or should have arranged for a second private sponsorship, the action was precluded by the discretionary function exception because the suit was essentially a challenge to United States immigration policies. And to the

extent the action was against the INS as a regulator of private conduct, the decision by the INS to release the Cuban refugee from the processing center was clearly precluded by the Supreme Court's pronouncement in *Varig, see* — U.S. at ——, 104 S.Ct. at 2764.

In another Fifth Circuit case the court dismissed a third-party complaint against an area manager of the National Weather Service for the failure to properly monitor weather conditions and supervise employees resulting in flood damage to the plaintiffs' property when, as a result, the (Louisiana) Parish Levee Board could not close flood gates in time. *Spencer v. New Orleans Levee Board*, 737 F.2d 435 (5th Cir. July 3, 1984). The court applied the discretionary function exception to dismiss the claim against the National Weather Service manager without analysis, citing a 1954 case holding that the federal Flood Control Acts immunized the United States and its employees from claims for property damage allegedly caused by inaccurate weather forecasts. *Id.* at 436. *Spencer* appears to have been decided based on a reluctance to hold the United States accountable in any manner for the vagaries of the weather; no analysis was presented on the extent to which acts or omissions implicated policy decisions as dictated by the Supreme Court in *Varig*.

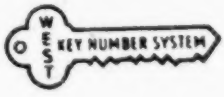
In *Begay v. United States*, 591 F.Supp. 991 (D.Ariz.1984), plaintiffs sought to hold various federal governmental agencies liable for failing to enforce safety levels at uranium mines. In an extensive discussion the court concluded that high-level governmental policy choices *directly* dictated the agencies' actions and, therefore, the discretionary function exception was applicable.

In the instant case, as previously stated, plaintiffs have alleged specific acts of negligence on the part of Dr. Baer, an employee of the federal government. The basic purpose of the FTCA is to make the United States liable for tort claims to the same extent and in the same manner as a private individual under similar circumstances. 28 U.S.C. § 2674. *United States v. Muniz*, 374 U.S. 150, 153, 83 S.Ct. 1850, 1853, 10 L.Ed.2d 805 (1963). Whether a claim is precluded by the discretionary function exception depends on the "nature and quality of the discretion involved in the acts complained of." *Indian Towing Co. v. United States*, 350 U.S. 61, 68, 76 S.Ct. 122, 126, 100 L.Ed. 48 (1955), *Smith v. United States*, 375 F.2d 243, 246 (5th Cir.1967). The fact that an individual actor employed by the federal government exercises *some* judgment in the carrying out of his responsibilities cannot be determinative, or the discretionary function exception would swallow the general rule permitting tort suits against the government. *See Caban v. United States*, 671 F.2d 1230, 1232 (2d Cir.1982); *Downs v. United States*, 522

F.2d 990, 995 (6th Cir.1975); *Griffin v. United States*, 500 F.2d 1059, 1063–64 (3rd Cir.1974). As in *Griffin*, the instant case in its present posture presents the issues of negligence in the exercise of professional and scientific judgment rather than policy-making and a concomitant balancing of competing considerations in determining the public interest. *See Griffin*, 500 F.2d at 1066. And, as in *Caban*, the activities of the defendant government employee, as the case currently stands, are not the kind of activities that involve weighing important policy choices, *see Caban*, 671 F.2d at 1233.

Accordingly, the motion of defendant United States to dismiss or for summary judgment is denied.

It is so Ordered.



Order of United States Court of Appeals for the Second Circuit Denying Petition for Rehearing, Dated March 4, 1991.

UNITED STATES COURT OF APPEALS

FOR THE SECOND CIRCUIT.

At a stated Term of the United States Court of Appeals for the Second Circuit, held at the United States Courthouse in the City of New York, on the 4th day of March one thousand nine hundred and ninety-one.

Present: Hon. George C. Pratt,
Hon. J. Daniel Mahoney,
Hon. John M. Walker, Jr.,
Circuit Judges,

JOANNA ANDRULONIS, Individually and as Conservator of the Property of JEROME ANDRULONIS,
Plaintiff-Appellees/Cross-Appellants,

against

UNITED STATES OF AMERICA, GLATT AIR TECHNIQUES, INC.; GLATT GMBH; WISCONSIN ALUMNI RESEARCH FOUNDATION, INC.; WARF INSTITUTE, INC.; RALTECH SCIENTIFIC SERVICES, INC., RALSTON PURINA COMPANY; ELI LILLY AND COMPANY; and JOHN L. THOMPSON AND SONS AND COMPANY,

Defendants,

UNITED STATES OF AMERICA,
Defendant-Appellant/Cross-Appellee.

Docket Nos. 89-6274, 90-6016, 90-6028

UNITED STATES OF AMERICA,
Third-Party Plaintiff-Appellee,

against

NEW YORK STATE DEPARTMENT OF HEALTH,
Third-Party Defendant-Appellant.

A petition for a rehearing having been filed herein by Third-Party Defendant-Appellant NEW YORK STATE DEPARTMENT OF HEALTH.

Upon consideration thereof, it is

Ordered that said petition be and it hereby is DENIED.

ELAINE B. GOLDSMITH,
Clerk
By TINA EVE BREE
Chief Deputy Clerk

Filed Mar. 4, 1991
Elaine B. Goldsmith, Clerk
United States Court of Appeals
Second Circuit

Order of United States Court of Appeals, Second Circuit, Dated January 28, 1991, Issued as Mandate March 12, 1991.

UNITED STATES COURT OF APPEALS

FOR THE SECOND CIRCUIT.

At a stated Term of the United States Court of Appeals for the Second Circuit, held at the United States Courthouse in the City of New York, on the twenty-eighth day of January one thousand nine hundred and ninety-one.

Present: Hon. George C. Pratt,
Hon. J. Daniel Mahoney,
Hon. John M. Walker, Jr.,
Circuit Judges,

JOANNA ANDRULONIS, Individually and as Conservator of the Property of JEROME ANDRULONIS,
Plaintiff-Appellees/Cross-Appellants,

against

UNITED STATES OF AMERICA, GLATT AIR TECHNIQUES, INC.; GLATT GMBH; WISCONSIN ALUMNI RESEARCH FOUNDATION, INC.; WARF INSTITUTE, INC.; RALTECH SCIENTIFIC SERVICES, INC., RALSTON PURINA COMPANY; ELI LILLY AND COMPANY; and JOHN L. THOMPSON AND SONS AND COMPANY,
Defendants,

UNITED STATES OF AMERICA,
Defendant-Appellant/Cross-Appellee.

Docket Nos. 89-6274, 90-6016, 90-6028

UNITED STATES OF AMERICA,
Third-Party Plaintiff-Appellee,

against

NEW YORK STATE DEPARTMENT OF HEALTH,
Third-Party Defendant-Appellant.

Appeal from the United States District Court for the Northern District of New York.

This cause came on to be heard on the transcript of record from the United States District Court for the Northern District of New York and was argued by counsel.

ON CONSIDERATION WHEREOF, it is now hereby ordered, adjudged and decreed that that portion of the judgment of the District Court that calculated the setoff under New York's General Obligations Law §15-108 from the parties own allocation of settlement proceeds is reversed and remand on that issue for further proceedings consistent with this opinion. In all other respects the judgment is affirmed.

ELAINE B. GOLDSMITH,
Clerk
By EDWARD J. GUARDARO
Deputy Clerk

Filed Jan. 28, 1991
Elaine B. Goldsmith, Clerk
United States Court of Appeals
Second Circuit

ISSUED AS MANDATE: March 12, 1991

Amended Judgment of the United States District Court for the Northern District of New York, Dated December 15, 1989.

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF NEW YORK

JOANNA ANDRULONIS *et al*

v.

UNITED STATES OF AMERICA *et al*

CASE NUMBER: 79CV847

Decision by Court. This action came to trial or hearing before the Court. The issues have been tried or heard and a decision has been rendered.

IT IS ORDERED AND ADJUDGED to enter an AMENDED JUDGMENT on the behalf of third-party Pltf United States against third-party defendant New York State Department of Health in the amount of $4,090,427, not $3,729,785 as originally directed by this Court.

Dated: December 15, 1989

J. R. SCULLY
Clerk

William Kaminska
(By) Deputy Clerk

Judgment of the United States District Court for the Northern District of New York, Dated October 18, 1989.

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF NEW YORK

JOANNA ANDRULONIS *et al*

v.

UNITED STATES OF AMERICA *et al*

CASE NUMBER: 79CV847

Decision by Court. This action came to trial or hearing before the Court. The issues have been tried or heard and a decision has been rendered.

IT IS ORDERED AND ADJUDGED in favor of Plaintiff Jerome Andrulonis against defendant United States of America in the amount of $5,978,409.00. Also judgment to be entered on behalf of Third-party plaintiff United States of America against third-party defendant New York State Department of Health in the amount of $3,729,785.00.

Dated: October 18, 1989

J. R. SCULLY
Clerk

William T. Kaminska
(By) Deputy Clerk